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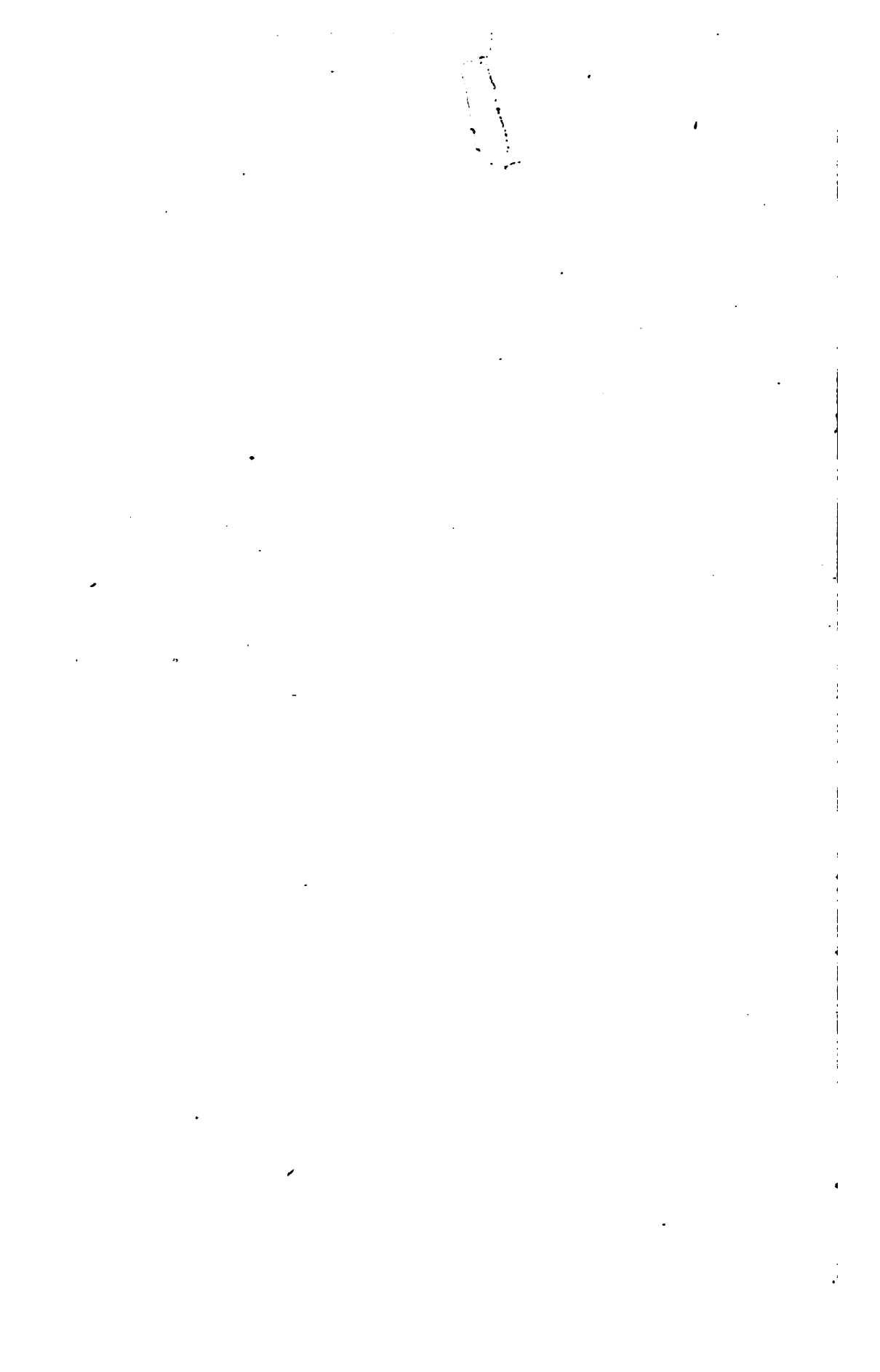
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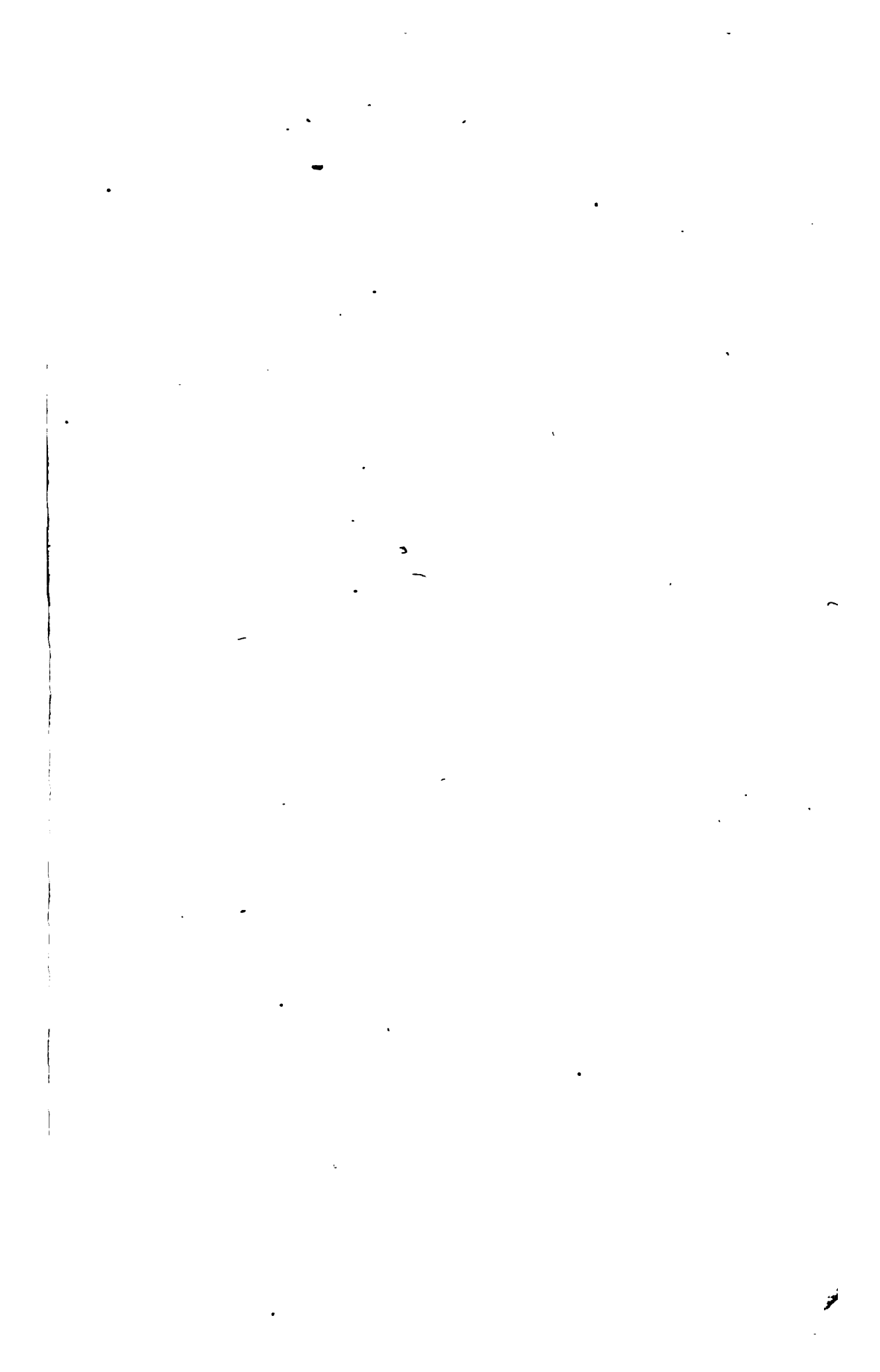
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AND
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EDITED BY

J. M'FADYEAN, M.B., B.Sc., F.R.S.E.

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PRICE 2s. 6d.

THE DISTRIBUTION OF THE LESIONS IN GENERAL-
ISED TUBERCULOSIS.

By J. M'FADYEAN, Royal Veterinary College, London.

IN dealing with cases of tuberculosis the term "generalised," ought to be reserved for those in which there are grounds for assuming that tubercle bacilli have been disseminated throughout the body by means of the blood stream. This is the sense in which the term is employed by all pathologists, but, unfortunately, there is still considerable divergence of opinion among authorities on meat inspection as to the interpretation which should be placed on the appearance and distribution of the lesions in determining in any given case whether generalisation has taken place or not. A view which appears to have obtained wide if not general acceptance in Germany and elsewhere on the Continent of Europe is that there are two different forms of generalisation in the ox—viz., acute and chronic. It is assumed that in the acute process a large number of tubercle bacilli are suddenly thrown into the blood stream, whereas in chronic generalisation there are smaller and perhaps repeated irruptions of the bacilli into the circulating blood. According to Ostertag,¹ who is one of the principal exponents of this view, acute generalisation is to be recognised at the *post-mortem* examination of an animal "by the presence of innumerable tubercles in most of the organs"; and the chronic process by the presence "of scattered nodules in various organs," especially in various groups of lymphatic glands. In an article which appeared in a former number of this *Journal*² I gave reasons for dissenting from this interpretation of the lesions found

¹ "Handbuch der Fleischschau," first edition, p. 392.

² Vol. XI., p. 226.

in natural cases of tuberculosis in cattle, and showed that the distribution of the tubercles in cases of experimental generalisation did not bear out the view that the irruption of large numbers of tubercle bacilli into the blood stream is followed by the development of innumerable tubercles in most of the organs. The purpose of the present article is to lead some further evidence of the same kind.

An opportunity to make observations with regard to the situation of the lesions which result from generalisation was afforded by a number of the experiments which were carried out at the Royal Veterinary College during 1899 and 1900, at the instance of the Committee appointed by the Royal Agricultural Society of England to obtain evidence concerning the reliability of the tuberculin test. In the Committee's Report¹ the lesions found at the *post-mortem* examination of each animal were stated briefly, but with sufficient detail to enable anyone to interpret the results of the experiment from the point of view of the inquiry—the reliability of the tuberculin test. In the following pages the particulars with regard to the autopsy of some of the animals will be set forth at greater length, and discussed from another point of view—viz., the evidence which they afford regarding the most constant distribution of the lesions in cases of generalised tuberculosis.

Experiment I.

The subject of this experiment was a calf nine months old. It was infected by injecting into the left jugular vein 3 cc. of liquid containing tubercle bacilli. The liquid had been prepared by mashing the entire spleen of a tuberculous rabbit with 12 cc. of bouillon, and allowing the coarser particles to fall to the bottom after the mixture had been poured into a test tube. The rabbit in question had been inoculated intravenously with tuberculous material from the spleen of a horse seventy-one days previously, and its spleen contained numerous tubercles. A cover-glass preparation made from the liquid used to inoculate the calf showed numerous tubercle bacilli, though they were not present in every field of the microscope ($\times 800$).

The calf was killed on the thirty-fourth day of the experiment, and the *post-mortem* examination revealed the following lesions:—

The lungs contained some thousands of grey tubercles about the size of dry sago grains. The bronchial and mediastinal lymphatic glands were enlarged to about four times the normal size, and on section they showed numerous spots and streaks of commencing caseation. No tubercles were visible in the liver, spleen, or kidneys.

Experiment II.

The subject of this experiment was a calf of about the same size and age as the one used in the preceding experiment. It was infected on the same day by injecting 3 cc. of the same liquid into the left jugular vein.

The calf was killed on the 100th day of the experiment, and the lesions found in it were as follows:—

Each lung contained a number of very firm, fibrous-looking tubercles; these were unequally distributed, but in each lung they

¹ See this Journal, Vol. XIII., p. 368.

were most numerous towards the base of the organ. The total number did not exceed a few dozens, and none of them was larger than a grain of barley. The pleura covering each lung carried a number of small fleshy growths, with the ordinary appearance of the tuberculous formations so commonly met with on the pleura in tuberculosis of the ox. The largest of these did not exceed the volume of a vetch pea, and had a distinct mushroom shape. In each lung they were most numerous on the diaphragmatic surface and the adjacent costal surface. The thoracic lymphatic glands were of normal size, and no tubercles were visible in any of them. The liver, spleen, and kidneys appeared to be free from tubercles, but one of the mesenteric glands contained a caseous and partially calcified nodule as large as a hazel nut.

It is scarcely possible to doubt that the last-mentioned lesion had been in existence before the date of the intravenous inoculation.

Tubercle bacilli could not be detected in cover-glass preparations from the pulmonary or pleural lesions, but a guinea-pig inoculated from one of the latter died with the lesions of a generalised tuberculosis after seventy days.

Experiment III.

The subject in this case was a yearling calf, and it was infected by injecting 1 cc. of liquid containing tubercle bacilli into the right jugular vein. The liquid was obtained by rubbing up a quantity of caseous matter from a tuberculous mesenteric gland of a horse with bouillon, and allowing the coarser particles to deposit. A cover-glass preparation from the liquid showed one or two tubercle bacilli in each field of the microscope.

The animal was killed on the thirtieth day after inoculation, and the following is the record of the *post-mortem* examination:—

Between the skin and the right jugular vein, at the seat of inoculation, there is a lesion about the size of a walnut. On section this is found to have a soft caseous centre. Scattered throughout the two lungs there are some scores of tubercles, about the size of barley grains. The centres of these are opaque from commencing necrosis and caseation. The bronchial and mediastinal glands are about thrice the normal size, but they show no distinct tubercles or caseation. No tubercles are visible in the liver, spleen, kidneys, or mesenteric glands. On the right side, one of the pharyngeal group of lymphatic glands is considerably enlarged and indurated, and on section it shows indications of commencing caseation. On the same side the prescapular lymphatic gland is double the normal size, and on section some grey tubercles are distinctly visible in its cortex. The left pharyngeal and prescapular glands are normal, and the following groups are normal on both sides: axillary, precrural, popliteal, and sublumbar.

Tubercle bacilli were detected in cover-glass preparations from the pulmonary tubercles, lesion over the jugular vein, and right pharyngeal and prescapular glands.

Experiment IV.

Subject, a calf of approximately the same size and age as the one used for the preceding experiment. It was inoculated on the same

day, and with the same material, but the quantity of fluid injected into the right jugular vein was $1\frac{1}{2}$ cc. It was killed forty-seven days after inoculation, and the following are the notes of the autopsy:—

Condition poor. In the upper third of the neck, over the course of the right jugular vein, there is a nodule somewhat larger than a garden bean. On section this shows some yellow tubercles. The prescapular lymphatic gland on the same side is three times as large as normal, and with the exception of the upper third it is visibly tuberculous, being greyish in colour, with streaks of caseation. A pharyngeal gland on the same side, close behind the hyoid bone, is walnut-sized and visibly caseating. Two other pharyngeal glands on the right side are in the same condition, one being nearly as large as a walnut and the other as large as a hazel nut. The prescapular and pharyngeal glands on the left side are normal in size and appearance. The pericardium contains about 3 ounces of clear, straw-coloured liquid. Serous membrane of pericardium normal. At several places on the left chest wall there is a slight fleshy villous growth (commencing serous tuberculosis). Pleural sacs contain two or three ounces of clear liquid. The bronchial and mediastinal glands are enormously enlarged. The left group is $3\frac{1}{2}$ inches in diameter; the right group is considerably larger. The mediastinal gland is about 8 inches in length, by about 3 inches in diameter at its thickest part. All these glands are grey on section, and present streaks and spots of caseation. Both lungs are almost uniformly filled with tubercles, varying in size from a vetch pea downwards. The tubercles are for the most part grey and opaque, but some are visibly caseous at the centre. Many of the lobules are in a state of collapse. The prepectoral glands are about the size of hazel nuts and apparently normal. The axillary glands on both sides are normal in size and appearance. Inguinal, precrural, and popliteal glands are normal on both sides. Peritoneum healthy. Two or three ounces of clear liquid in the sac. The hepatic lymphatic glands are normal in size, but contain some visible tubercles. The liver contains some scores of tubercles, scarcely larger than pin's heads. The spleen contains one tubercle, about the size of a barley grain. One kidney contains a tubercle as large as a mustard seed. Other abdominal lymphatic glands normal. Muscular masses of hind limb sliced up and searched for tubercles, but none found.

Cover-glass preparations were made from the splenic and renal tubercles and from the pleural growth, and tubercle bacilli were detected in the first and last of these, but not in the tubercle of the kidney.

Experiment V.

Subject, a calf similar in size, age, and breed to the animals used in the preceding two, and the following two, experiments. The conditions of the experiment were the same as in the preceding two cases, except that the quantity of liquid injected into the right jugular vein was 2 cc. The animal was killed on the fifty-fourth day after inoculation, and the following are the notes of the *post-mortem* examination:—

Condition fair. At the seat of inoculation, over the course of the right jugular, a nodule about the size of a garden bean is

adherent to the sheath of the vein. On section this is visibly caseating. The lowest of the pharyngeal glands above this on the same side is visibly caseous, and twice as large as its fellow on the opposite side. A gland a little in front of this is larger than its fellow, but not visibly diseased. The following groups of glands are normal in size and appearance: prescapular, axillary, precrural, popliteal, and abdominal glands generally. The lungs are uniformly filled with firm tubercles, varying in size from a vetch pea to a barley grain. On section some of these are grey and others are opaque and caseating at the centre. At several places the pleura carries small fleshy, mushroom-like growths. Bronchial and mediastinal glands are enlarged to three or four times above the normal, and on section they show streaks of caseation. Peritoneum normal. Kidneys normal, save for one pinhead, white tubercle in one of them. Spleen normal. About a dozen white tubercles, about the size of small barley grains, in the liver. No tubercles visible in the muscular masses of fore or hind limbs.

Tubercle bacilli found in liver tubercles, but not in kidney tubercle.

Experiment VI.

This experiment was an exact parallel to the preceding one, the calf being of approximately the same size and age, and all the conditions identical.

The calf was killed on the 106th day after inoculation, and the autopsy revealed the following lesions:—

Subcutaneously over the right jugular, at the seat of inoculation, there is a nodule as large as a hazel nut, with a fibrous capsule enclosing thick yellow caseo-pus. In the right lung there is present one tubercle about the size of a barley grain, with soft caseo-purulent contents. Towards the base of each lung there are some scattered, firm, fibrous tubercles, without any caseous centres. The bronchial and mediastinal glands are a trifle larger than normal, and all contain a few yellow tubercles, varying in size from a pin's head downwards. Kidneys, liver, and spleen normal. Pharyngeal lymphatic glands symmetrical on the two sides, and show no tubercles or degeneration. Mesenteric glands normal. Right prescapular gland normal in size, but contains two tubercles with soft caseous contents, the largest the size of a vetch pea. Other body groups of lymphatic glands normal. Heart normal.

Tubercle bacilli found in tubercle of prescapular lymphatic gland, lesion over jugular, caseous large lung tubercle, and tubercle of the mediastinal gland. Other lesions not examined for bacilli.

Experiment VII.

This experiment was exactly parallel to Experiment IV., except that the animal was not killed till the 106th day after inoculation. The lesions found in it were as follows:—

No lesion at seat of inoculation. Bronchial and mediastinal glands quite normal in size and appearance on section. At the base of the left lung, close to its diaphragmatic edge, there are a few firm, shotty, fibrous tubercles. Lungs otherwise normal. Spleen, liver, and kidneys normal. Tongue and throat and pharyngeal lymphatic

glands normal. Mesenteric glands normal. All the groups of body lymphatic glands normal. Heart normal.

Experiment VIII.

The animals used for this and the next experiment were yearling cattle, and the method of infection employed was the intra-venous injection of 2 cc. of a liquid rich in tubercle bacilli, the vessel selected being the right jugular vein. The liquid was prepared as follows. A quantity of caseo-purulent matter from the tuberculous spleen of a horse (about half the volume of a common pea) was rubbed up in a mortar with 10 cc. of bouillon, and the coarser particles were allowed to settle. A cover-glass preparation from the liquid showed that it was very rich in tubercle bacilli, some being present in every field of the microscope. The liquid was therefore further diluted with an equal volume of bouillon. As the results in some of the previous experiments indicated that a trace of the liquid had escaped into the connective tissue between the vein and the skin, probably in the act of withdrawing the needle of the syringe, an attempt was made to guard against this by sucking a quantity of blood out of the vein into the syringe before withdrawing the needle, the object being to thus wash away any tubercle bacilli that would otherwise have been left at the extremity of the needle.

The animal was killed on the sixty-fourth day after inoculation, and the following are the notes of the *post-mortem* examination:—

There is no lesion at the point of inoculation in the neck. The entire lung tissue is crammed with miliary tubercles, which are grey and not visibly caseous. The lung tissue appears to be most thickly beset with tubercles in the upper part of each organ, under the costo-vertebral edge of the lung. At some places in this position the lung tissue is solid from confluence of the tubercles. The bronchial and mediastinal lymphatic glands are enlarged to three or four times the normal size; the gland tissue is much congested, and without exception the cortex is to a large extent caseous, and perceptibly gritty from calcification. The pleura is normal. With the exception of some doubtful specks in a few of the mesenteric glands, the other groups of lymphatic glands are normal in size and appearance. No tubercles are visible in the heart, liver, or spleen, but a few of the size of mustard seeds are present in the kidneys. The peritoneum is healthy.

Experiment IX.

All the conditions of this experiment were identical with those of the preceding one, and the animal was also killed on the sixty-fourth day after inoculation. The following are the lesions that were noted at the *post-mortem* examination:—

The lung tissue contains scattered pin-head firm tubercles (less numerous than in the previous case). The bronchial and mediastinal glands are all about three times the normal size, extensively caseated, and slightly gritty. A small number of tubercles about the size of mustard seeds are present in the substance of the liver, but none are visible in the kidneys, spleen, or heart. The pleura and peritoneum, and all the groups of lymphatic glands, except those mentioned, appear normal. There is no lesion at the seat of inoculation in the neck.

Experiment X.

The subject of this experiment was an eighteen-months-old heifer, which had been purchased for the tuberculin inquiry four months previously, but was not used for that series of experiments because it reacted to the preliminary test with tuberculin. For the purpose of the present experiment it was infected by injecting $1\frac{1}{2}$ cc. of liquid containing tubercle bacilli into the left jugular vein. The liquid was obtained by rubbing up a portion of the tuberculous liver of a rabbit (infected by intra-venous inoculation) with a quantity of bouillon, and allowing the coarser particles to settle.

The animal was killed on the fifty-first day after inoculation, and the lesions discovered at the *post-mortem* examination were as follows :—

About the middle of the neck, adherent to the left jugular, there is a greyish growth about three-quarters of an inch in diameter and a quarter of an inch in maximum thickness ; on section it shows some minute yellow points. There are some easily destroyed adhesions between the left lung and the chest wall, mediastinum, and diaphragm. At some places on the diaphragm and chest wall there are small patches of grapy tubercles, caseous and partly calcified. There is also, between these patches, a pinkish, soft, diffuse growth. There is no liquid in the left pleural sac. The pericardium contains about 4 ounces of clear watery liquid. The right pleura shows lesions identical with the left. No liquid in the sac. The bronchial glands are enlarged, the largest one on the right side being about the size of a pigeon's egg ; it is almost entirely stony in consistence from calcification. The largest gland on the opposite side is about the same size, and it contains several completely calcified nodules. The lung tissue is almost uniformly filled with pin-head fibrous-looking tubercles, giving a shotty feeling to the lung when it is grasped. The mediastinal gland is normal. The peritoneum contains about an ounce of clear watery liquid. There are some adhesions between the rumen and the spleen on the one hand and the abdominal wall on the other. On the capsule of the spleen and on the exterior of the rumen there are some yellow calcified tubercles. Several of the mesenteric glands contain calcified nodules. There are some adhesions between the liver and diaphragm, and numerous grapy growths on the opposed surfaces. No tubercles are visible in the parenchyma of the liver, spleen, or kidneys. The left popliteal gland is normal in size, but in its substance there is a calcified tubercle about the size of a barley grain. The lymphatic glands of the head and throat are normal ; also on both sides the prepectoral, pre-scapular, axillary, precrural, pelvic, sublumbar, and supra-mammary. No lesions discoverable in the muscular masses of the body.

The results of this series of ten experiments may now be analysed in order to see what support the results lend to the view that the entrance of tubercle bacilli into the blood stream is followed by an eruption of innumerable tubercles in all the organs, and notably in the spleen and lymphatic glands. It may be well to deal in the first place with the four important organs—lungs, liver, spleen, and kidneys, and the results so far as these are concerned are summarised

in the subjoined table, in which the sign + indicates that the organ in question contained tubercles, and — that it appeared to be free from tubercles.

<i>Experiment.</i>	<i>Lungs.</i>	<i>Liver.</i>	<i>Spleen.</i>	<i>Kidneys.</i>
No. 1.	+	—	—	—
„ 2.	+	—	—	—
„ 3.	+	—	—	—
„ 4.	+	+	+	+
„ 5.	+	+	—	+
„ 6.	+	—	—	—
„ 7.	+	—	—	—
„ 8.	+	—	—	+
„ 9.	+	+	—	—
„ 10.	+	—	—	—

It will be observed from this table that in each of the ten experiments the lung tissue contained tubercles the appearance of which was in harmony with the view that they were embolic in their origin, and ascribable to the arrest in the pulmonary capillaries of some of the tubercle bacilli experimentally introduced into the blood stream. Similar lesions were present in the liver and kidneys in only three of the cases, and in the spleen in only one. As a matter of fact it is very doubtful whether the minute lesion which was present in one of the kidneys in Experiments IV. and V. was a true tubercle. In each of these cases the entire renal tubercle was crushed and stained for tubercle bacilli, and with a negative result.

It is, of course, not pretended that the macroscopic examination of the liver, spleen, and kidneys, although it was made with great care, entitles one to assert that the organs mentioned were free from tubercles in the cases in which none were found; even the negative result of a very prolonged microscopic examination of sections prepared from the organs would not have entirely justified such a conclusion, but, at the same time, in view of the period which had elapsed between infection and death in most of the cases, and the considerable size which the pulmonary tubercles had attained, it is impossible to deny that the experiments amount to very strong evidence against the view that, in the ox, the irruption of large numbers of tubercle bacilli into the blood stream is followed by the development of numerous tubercles in all the internal organs, and notably in the spleen. It is not improbable that the result might have been different had a very much larger number of tubercle bacilli been injected into

the veins, but it will hardly be contended by anyone that there is any reason to suppose that a much larger number of the bacilli are ever thrown into the blood stream in natural cases of generalisation. At any rate, it has never been my lot to see a natural case of generalised tuberculosis in any animal with a denser crop of tubercles in the lung tissue than was present in a number of these experimental cases.

Coming next to the question of the participation of the lymphatic glands in the arrest of tubercle bacilli from the circulating blood, an analysis of the results brings out some very interesting points, but lends no support to the theory that the presence or absence of tubercles from various groups of peripheral lymphatic glands furnishes a criterion from which one may judge whether in any given case generalisation has taken place or not.

In this connection Experiment X. must be separated from the others, as in that case the animal was undoubtedly the subject of a previous natural infection. Putting that case aside for the moment, it will be found that tuberculous lesions were present in what may be called peripheral groups of glands in four of the remaining nine experiments. In Experiments II. and IV. such lesions were present in the pharyngeal and prescapular groups on the right side of the body; in Experiment V. the right pharyngeal group was involved, and in Experiment VII. the right prescapular gland was similarly involved. Since in each of these cases the animal had failed to react to tuberculin before it was experimentally infected, the development of lesions in the above-mentioned groups of glands might at first sight be interpreted as evidence in favour of the view that the lesions frequently found in these and other peripheral groups of glands in natural cases of tuberculosis are embolic in their origin, but closer examination compels another interpretation. In each of these four cases there was a tuberculous lesion at the seat of inoculation, obviously due to the escape of a trace of the infective liquid into the connective tissue in withdrawing the needle from the vein. It would be quite unreasonable to explain as a mere accident or coincidence the unilateral situation of the glandular lesions, and the fact that in each case these were on the same side as the lesion at the seat of inoculation. The escape of liquid into the connective tissue at the seat of inoculation was accidental and undesired, but there is no reason to regret its occurrence, since it furnished very interesting evidence with regard to the method of infection of the lymphatic glands in cases of tuberculosis.

The only reasonable explanation of the genesis of the lesions in the pharyngeal and prescapular glands is that the infection took place by way of the lymphatics from the lesion over the jugular vein, at the point of inoculation. Probably no one will see any difficulty in the way of accepting this explanation of the lesions in the prescapular group, but some may hesitate to admit that the pharyngeal group were also invaded by the bacilli by way of the lymphatic vessels, since that implies an extension of the disease contrary to what is generally supposed to be the ordinary course of the stream of lymph. Nevertheless, it is, as already said, the only reasonable explanation of the facts, for it would be too fantastic to suppose that in each of these four cases tubercle bacilli circulating with the blood

were in some mysterious way arrested in the groups of glands nearest to the point of inoculation, and in none of the other peripheral groups of glands.

It may be of interest here to recall the fact that in one of the series of three experiments of the same kind, of which an account has already been published,¹ tubercles were also present in the pharyngeal and prescapular glands on the side on which the animal had been inoculated into the jugular. In that case, however, the point of inoculation was not examined at the *post-mortem* examination, and what now appears to me to be the correct inference with regard to the method of infection of the glands was not drawn.

The only other case in which a peripheral lymphatic gland was found to be the seat of tuberculous disease was Experiment X., in which a calcified tubercle was present in one of the popliteal glands. But, as previously mentioned, that animal had reacted to tuberculin four months before it was infected by intra-venous inoculation, and it appears to be possible, and even probable that, with the exception of the miliary tuberculosis of the lungs and the nodule over the sheath of the jugular vein, all the lesions found at the *post-mortem* examination had been in existence before the animal was experimentally infected.

The experiments thus indicate that in the ox, even when large numbers of tubercle bacilli are thrown into the blood stream, embolic tubercles in the lymphatic glands are not thereby set up. Such, at any rate, appears to be the rule, though it is conceivable that exceptions might occur if very much larger numbers of bacilli than were used in these experiments were suddenly introduced into the blood. But this admission does not weaken the contention that in practice embolic tubercles in the lymphatic glands of the ox must be rare, for, as previously remarked, it is improbable that very much larger numbers of bacilli than were employed in the most of these experiments ever find their way into the blood stream in natural cases of generalisation. If they did, it may confidently be asserted that the resulting pulmonary tubercles would be so numerous as to determine the animal's death within a very short period—a period too short to allow of one explaining in this way the presence in the lymphatic glands of large macroscopic lesions.

But, as previously mentioned, the view has been put forward that it is mainly the irruption of small numbers of tubercle bacilli into the blood stream that is followed by the development of embolic tubercles in various groups of lymphatic glands. If it is admitted that as a rule such lesions are not set up in the lymphatic glands even when enormous numbers of bacilli are suddenly thrown into the blood, this theory hardly appears to require serious refutation. One cannot imagine any *a priori* argument in favour of it, and it is as yet entirely unsupported by experiment. It is simply an improbable hypothesis which has been evolved by those who have failed to recognise that any of the lymphatic glands of the body may be infected in a centrifugal direction by way of the lymphatic vessels from a tuberculous lesion in their neighbourhood.

The results of these experiments, so far as they bear on the methods by which tuberculous disease is spread within the body,

¹ Vol. XI., p. 245.

entirely corroborate the conclusions placed at the end of the former article to which reference has already been made, viz. :—

1. The never failing evidence of generalisation is the presence of tubercles of approximately equal size scattered throughout the entire spongy lung tissue.

2. In the absence of the lungs, the other organs and the carcase generally do not afford to a naked-eye examination reliable evidence as to whether generalisation has taken place or not.

3. In cases of generalised tuberculosis either the kidneys, liver, or spleen, or the whole of these at once, may be free from macroscopic lesions although the lungs contain myriads of embolic tubercles visible to the naked eye.

4. The macroscopic tubercles which are met with in the liver, spleen, kidneys, and lymphatic glands generally are as a rule due to lymphatic infection, and afford no evidence of generalisation by way of the blood stream.

The present series of experiments also warrants the following further conclusion :—

5. In at least some parts of the body tubercle bacilli are readily carried in the lymphatic vessels in a centrifugal direction, that is to say, in a direction contrary to that in which the lymph stream is generally supposed to flow in normal circumstances.

These conclusions do not apply to the interpretation of the lesions in cases of congenital tuberculosis, in which the distribution of the tubercle bacilli throughout the body is modified by the peculiarities in the foetal circulation.¹

Finally, a number of the experiments described in this article have yielded results that are remarkable from another point of view than the one that has been specially considered in the preceding pages. The absence of uniformity in the results of experiments that were identical as regards the method of infection and the amount of the infective material employed cannot have escaped the reader's notice. A no less striking feature of some of the experiments is the evidence of a process of recovery which was disclosed by the *post-mortem* examination. Comment upon these aspects of the results must be deferred until another occasion.

THE POST-MORTEM DIAGNOSIS OF ANTHRAX IN CATTLE.

By H. CAULTON REEKS, F.R.C.V.S., Spalding.

ONE would imagine, taking into consideration the teeming literature on the subject of anthrax in cattle, that little more need be written concerning its *post-mortem* diagnosis. My only excuse for adding still further to the amount of matter already published is that the question is an all-important one to the country veterinary surgeon, more especially if he be constantly employed by the Local Authority of his district as their inspector.

¹ See Vol. XII. of this Journal, p. 156.

Assuming a case of suspected anthrax to have been reported to him, this is briefly the quandary in which he finds himself:—

- (a) Shall he, from mere outside appearances, give an opinion off-hand as to the cause of death?
- (b) Shall he make a *post-mortem*?
- (c) Shall he, not being an expert microscopist, base his diagnosis largely on the microscopic examination of the blood?
- (d) Shall he send a sample of the blood or spleen to an expert, and await his reply?

We will consider each of these questions in the order here named, and see what conclusion we arrive at.

(a) Is it wise to give an opinion off-hand, ignoring the use of the microscope, and dispensing with the *post-mortem* as dangerous? It cannot be denied that this is the readiest method—one which will recommend itself to the lazily inclined. That it is the most likely to give a trustworthy result, however, is not so apparent. Deaths from other causes may so closely simulate, in outward *post-mortem* appearances, death from anthrax, as to render it positively foolhardy to attempt to give in this manner a true verdict of the cause of decease. Notably is this the case with death from lightning stroke. In both cases there will be rapid putrefaction of the carcase; in both cases the escaping blood will be abnormally dark, tarry, and fluid; in both cases there will be frothy, blood-stained, discharge from the nostrils and rectum; and in both cases the last-named organ may, or may not, be found everted.

The veterinarian who attempts to give his diagnosis and base his conclusions solely on this old-fashioned, primitive, and cursory method of examination will sometimes be likely—nay, more than likely, sure—to fall into one of these two errors. First, a death from any other cause than anthrax he may return in the positive, consoling himself with the thought that if it is an error it is one on the safe side, but forgetting in his anxiety to make his own way smooth that he will put the owner, possibly a client, to unnecessary trouble and inconvenience, and probably even direct monetary loss. Secondly, it is quite upon the boards that the veterinarian may make a graver mistake still. He may probably return as “a case of no importance” one of anthrax. I need hardly say, in a journal of this description, that a mistake of that nature would be fraught with danger to all who afterwards handled the carcase—direct danger to human life. That is leaving out of the question altogether the terrible risk that the owner will run of further infecting his pastures or buildings, and the loss he may incur from death of other stock.

I am quite prepared to admit that in ninety-nine cases out of every hundred a man might by this means hit the right nail on the head when giving his diagnosis. To my mind, however, that offers no excuse for not taking other, and more advanced, methods into his employ.

(b) Shall a *post-mortem* be made? Undoubtedly everyone will answer “No!” Unless extremely careful, or extremely fortunate, the surgeon, or the men assisting him, may become inoculated with the virulent material, and a case of malignant pustule, perchance fatal, is the result. Short as has been the time I have been in practice, I have seen loss of human life from this too frequent and

absurd practice. Too absurd to countenance for even one moment. Absurd to a point verging on criminality. Again, the remarks with regard to the infecting of the land and the further spreading of the disease will apply here, so that, concerning the question of making a *post-mortem*, we may wind up emphatically "Do not!"

(c) Shall he, not being an expert microscopist, base his diagnosis mainly on the microscopic examination of a stained and mounted specimen of the blood? To this question I shall give another emphatic answer, this time in the affirmative. I maintain that this is the only way left to the veterinarian to arrive at a just conclusion within a reasonable time. I am prepared to go even a step farther than that. It is quite possible, I think quite as reliable, to base a diagnosis on an inspection of the blood unstained—simply a droplet mounted under a cover on a slip.

Having examined the carcase, taken due note of all outside circumstances, learned the history of the case, etc., we come mentally to one of these two conclusions: either it is anthrax, or it is not anthrax. We do not, however, at this stage express our opinion to the owner.

So far, we have diagnosed the case off-hand, and are quite as likely to be right as any other man who adopts that method. Now, however, we are going to do better than he, by calling to our assistance a more delicate means of diagnosis than the unaided senses. We cut off one of the ears and then proceed as follows, asking ourselves the undermentioned questions, and giving our own answers:—

How long has the animal been dead? Well, within twenty-four or thirty hours we have been told. In that case, we reply, we may, if the animal is dead of anthrax, expect to find anthrax bacilli in the blood. If, on the other hand, we are able to assure ourselves that death has occurred longer than thirty-six to forty-eight hours, we may begin to look upon the finding of anthrax bacilli in the blood as doubtful, unless the weather is that of winter. If life has been extinct longer than three days, no matter what the weather, we may confidently assert before commencing a search that nothing will be found in the blood. Rather, I should have said, something will be found, but it will not be anthrax.

Is the blood darker than normal, more tarry in appearance, and more fluid? Yes. Then it may be anthrax. No. Then it may not be.

Is there any smell beyond that of ordinary fresh blood? Is there any foetor? In other words, is putrefaction going on? If putrefaction has set in, we reply to ourselves, then although the animal may have died of anthrax we need not expect to find anthrax bacilli in the blood. The reason every tyro in bacteriology knows full well. The putrefactive organisms from the intestine, taking advantage of the deoxygenated state of the blood, have multiplied at an enormous rate, and completely routed the anthrax bacilli from their former proud position. If distinct evidence of putrefaction exists, as revealed by smell, then we are not, no matter what we see, to call any bacillus we may light upon in an unstained specimen that of anthrax.

If, on the other hand, every other circumstance being in accordance—blood tarry, dark, and fluid; animal only recently dead; putrefaction absent—we find hosts of a rod-shaped organism in the blood, we may decidedly declare that the bacilli in question are those of

anthrax. The owner may then, with every assurance of our correctness, be warned of the nature of his case, and the Local Authorities may have the information immediately sent in to them. The case is out of the veterinarian's hands. If putrefaction has set in, and it is impossible to find anthrax bacilli under the cover glass, then it simply remains that the carcase must be buried intact under suspicion. That is, of course, if other circumstances are, to use a slang term, "fishy." In any case the veterinary surgeon is no whit the worse for having attempted the search. Very often he is much to the good.

We may sum up in this manner. Provided the bacilli of anthrax are present in the blood, and we have a microscope magnifying only 400 diameters, we stand as fair a chance of seeing what we are looking for as the expert.

If the bacilli, from degeneration or other cause, are absent from the blood, and spores of anthrax are presumed to be present, is there then any method available for their determination? Yes, two. One, physiological separation, the veterinarian cannot adopt owing to the state of the vivisection laws. The other, cultivation, he may.

Here, I may remark, the anthrax bacillus is one that lends itself with comparative ease to artificial growth. Any veterinarian who has been through a modern course of bacteriology—say, has finished his course at the Royal Veterinary College during the last seven or eight years—will have no difficulty in searching for, and, if needful, cultivating the bacillus of anthrax, experts saying what they will to the contrary. I have done it myself, and I do not lay any claims to exceptional ability. Any other man, with a facility for common sense reasoning and the application of what he has learned at college, may do the same. As I have remarked before, the bacillus anthracis is comparatively easy to grow. Also, it is the largest of the important pathogenic rods; it has the most known of its morphology, life history, etc.; and it is the one the veterinary surgeon will come into frequent contact with. Sterilised test-tubes of bouillon are easily made; the method of inoculating a successive series of the same is adopted. If an incubator, with its properly regulated thermometer, etc., is not at hand, then the temperature of most kitchen mantleshelves will answer admirably to coax their growth; and in two or three days' time one, perhaps two, of the tubes will reveal a first-class growth of clean, feathery-looking anthrax. A particle placed on a slide, stained, and mounted will, under the microscope, convince anyone of its real nature.

To be correct, the test ought to be carried a step or two further. A small animal should be inoculated, and the bacillus again obtained from its blood. There is, however, no necessity for the veterinarian to do that. If an animal of the bovine species is found dead under circumstances suspicious of anthrax; if from the cultivation of that blood in a test tube a material is produced exactly resembling anthrax; and if a particle of that growth on a slide, when suitably stained and mounted, reveals interlacing leptothrix filaments, not even the expert would care to dispute the fact of its certainly being anthrax.

In any case, should he have deemed surrounding circumstances such as to warrant him in searching further for anthrax, the veterinarian will have had the carcase properly put away, and will have

communicated with the police. Further search is simply that he may have by him other and irrefutable proof of the correctness of his diagnosis should occasion arise to demand it. Also, that he may further perfect his knowledge of scientific methods of search.

Concerning an attempt to come into close quarters with any other pathogenic bacillus, the expert bacteriologist may, with some degree of foundation for his caution, warn the tyro off. With anthrax, however, he has taken the wrong bull by the horns, and is carrying too high a hand.

(d) Shall I send a sample of the blood or spleen to an expert, and await his reply? First, take the advantages of this method. The expert, with his full and better means of conducting an investigation, with the knowledge of many years' experience, and with the dexterity born of long practice, will be far more likely to arrive at a right result than the newly-fledged M.R.C.V.S. The expert, however, must have had a beginning, and my advice to the newly-made M.R.C.V.S. is this: During your first two or three years' practice avail yourself of the expert's advice. While so doing, however, take advantage of the means at your disposal for acquiring some measure of the expert's knowledge.

Another advantage of this method is, once you have sent off the specimen, you are relieved of all further anxiety and responsibility. This, in my opinion, should not weigh with any man anxious to improve his own knowledge and to advance with the times,

Taking the disadvantages of this method into full consideration, it will be found that they weigh far too heavily to ever recommend a practitioner to follow it in every case.

In the first place, if we are to rely on the expert's advice, there will be a great deal of time wasted between the forwarding of the specimen by post and the receiving of the expert's decision. This, with the police waiting to put disinfectant measures into operation, will be irritating to both parties.

In the second place, what is to be done with the carcase during this investigation? Disinfect, and bury under suspicion? If we do that, and the expert's decision is "Not anthrax," we are in hot water with the authorities for putting the county to unnecessary expense, and are in the black books of our client for subjecting him to needless inconvenience and loss.

Should the carcase be preserved intact pending the expert's investigations? That, should the animal really have died of anthrax, would be next to criminal—certainly not to be advised.

Thirdly, is there not a chance, and that not a remote one, of the expert returning a false verdict? Undoubtedly there is a chance of his so doing, and, to my mind, that constitutes the greatest obstacle in the way of adopting this method of arriving at a correct diagnosis. There are frequently cases of anthrax in which, if the carcase has lain long with the temperature at summer heat, the bacilli have totally disappeared. Not only have they disappeared, but they have become absolutely destroyed. As we have every reason to believe that the bacillus anthracis does not sporulate within the blood stream, it follows that there is absence of both bacillus and spores.

A sample of this—I may call it for the moment "barren blood"—is forwarded to the expert. He, like the Israelites of old, cannot

make bricks without straw. He cannot demonstrate anthrax bacilli where anthrax bacilli do not exist. He cannot cultivate anthrax bacilli from a material barren alike in germs and in spores.

He is compelled, from the very nature of the evidence he obtains, to return the case as "Not anthrax." He makes a mistake. Acting upon his advice, the carcase is not disinfected in the usual manner—is not, at any rate, disposed of so carefully as it should have been. Portions of the evacuated blood, fæces, etc., will be left to further infect the ground.

In fine, the expert has made as serious a blunder as the practitioner unaided might have done.

Finally, my advice to the country veterinarian is: Use your own God-given powers of observation, supplement them by the use of a good microscope, and give your verdict freely and fearlessly. I do not think you will often fall into serious error.

FOOT-AND-MOUTH DISEASE.

By J. PENBERTHY, F.R.C.V.S., Royal Veterinary College, London.

That we should desire to be conversant with so important a subject needs no apology, while the great agricultural community, which it is our privilege to serve, will not underrate any evidence of our interest in a matter so materially affecting their own, and in connection with which we, as a profession, may justly claim to have heretofore rendered service of immense value.

To the younger members of the profession there may be something of novelty connected with the subject, for while those of us whose professional experience extends over two decades will readily recall a time when foot-and-mouth disease appeared to be like the poor, "ever with us," many of the rising generation of veterinary surgeons, and of stock-keepers too, cannot possibly have had any personal acquaintance with the affection. Recent events have aroused fresh interest in the disease, and clearly indicate that, even, in its absence, it behoves all of us to be able to recognise and deal promptly with it should occasion require, also that "like a thief in the night" it is liable to come and make its presence felt when least expected. In consideration of these circumstances I must crave the indulgence of the older members if in course of my remarks I at times dwell in detail on points with which they are familiar.

Though I have no intention of attempting to exhaust this part of the subject, it may be well, in order that its importance be realised, to look backwards and briefly glance at the history of foot-and-mouth disease. Reference to the older writers on veterinary and agricultural matters might lead us to the conclusion that the disease prevailed in England as far back as the middle of the seventeenth century, but there is little definitely known of its existence in this country till 1839. Indeed, whether or not it had *ever* existed here at any antecedent date, it would appear that for many preceding years it was exotic, as agricultural and veterinary literature of the earlier part of this century is silent on the point.

The records of the period tell us that in 1834 the disease was

prevalent in southern Europe; in 1837 it raged in Switzerland, and in 1839 it was widespread over France and Holland. By the latter part of 1839 it had reached England, Scotland, and Ireland. In this connection it is curious to note that the very first order (3rd and 4th William IV. cap. 52) prohibiting the landing of foreign cattle, sheep, and swine in England and Ireland was in force from 1833 to 1842. The exact means by which the disease was introduced at this time has never been conclusively demonstrated, and the circumstance did not tend to convince the popular mind of its contagious nature, but rather appeared to give support to the convenient and prevailing idea of "spontaneous generation." There can be little wonder that at a period when so little was known of the nature of contagious diseases, the above view was strongly expressed in argument against restrictions as to landing foreign animals and movement—indeed, against all irksome regulations. As indicated by veterinary surgeons at the time, a possible and probable source of the disease existed in those animals, cows, sheep, and pigs, which almost invariably formed part of every ship's stores, and which it was permissible to land after inspection by a veterinary officer, at that time associated with the Customs. Importation of hides, etc., was also blamed.

The credit of first recognising the disease in England belongs to an Eastern Counties' man, of whose association with cattle pathology we are all justly proud, to whom we readily accede the position of "father" of that branch of our art, and to whose acumen and perseverance the stock-owning community, as well as the veterinary profession, must ever remain deeply indebted. In 1839 Professor Simonds identified the disease in a herd of cattle grazing at Laleham, near Twickenham, in Surrey, where he then practised. After this identification it was soon realised that cases of the disease had previously occurred in Smithfield market, while almost simultaneously its existence in the great market of Norwich was recorded, and thus was initiated an experience for Norfolk which continued to grow, and gain for the county a notoriety in this connection far from enviable. It must, however, be admitted that the experience of Norfolk was not without favourable influence, for if Norwich market became notorious as a centre from which foot-and-mouth disease appeared to spread throughout the length and breadth of the land, the lessons of acute and intimate experience did not fail to arrest the powers of observation, and arouse the spirit of determination of advanced agriculturists of Norfolk. The history of the disease in Great Britain recalls the invaluable service rendered by such men as Mr Clare Sewell Read and others in supporting against rabid opposition the veterinary demand for "short and decisive action" in dealing with this and other diseases.

However introduced, the affection remained with us, marking decline or disappearance, and recrudescence or recurrence, from 1839 to 1886. Up to 1870 there are no reliable records as to the number of outbreaks or animals affected, but the current veterinary publications tell us very plainly that again and again the disease spread over the country, exhausted its virulence or its pabulum, and after a while again broke out.

In 1842 the prohibition order was cancelled, and an import duty imposed, 20s. on each head of cattle, and 1s. on each head of sheep and

swine. During the existence of this impost the country appeared to remain almost if not altogether free. In 1846 this duty was removed, and by 1852 foot-and-mouth disease became widespread in England, Scotland, and Ireland. By 1861 it had declined to very narrow limits, but soon it again began to increase, and by the time that cattle plague appeared in 1865 it was general. In this year the legislature made their first attempt to systematically control contagious animal diseases. The veterinary department of the Privy Council was now formed, cattle plague was extirpated, and some confidence was manifested in veterinary opinion. Coincident with the extirpation of cattle plague was a decline of foot-and-mouth disease. In 1864 an attempt was made to include this affection in a bill brought before Parliament by Dr Bruce and Sir George Grey. This was violently opposed by agriculturists, who appeared to dread the restrictions more than the disease, and it was not until after the suppression of cattle plague, namely in 1869, that this disease was scheduled. At first all orders were of a permissive character, giving Local Authorities the power to act if they thought fit. It was not till 1878 that the regulation became compulsory, and, in addition to possessing the powers, Local Authorities were forced to exercise them. The Central Authority recognised the great importance of preventing introduction of the contagion with foreign animals, and the Privy Council issued an order prohibiting the landing of any cattle, sheep, or swine from countries in which foot-and-mouth disease was known to exist, and also exercised through a Central Authority the delimitation of infected areas and the restriction of movement.

As indicating in some degree the tendency of foot-and-mouth disease to spread, and the consequent loss and annoyance, it will be enough to refer to the fact that, notwithstanding the great decline up to 1869, in 1870 the disease was reported in ninety-two counties, 52,164 outbreaks were noted, and 691,560 animals attacked. Though no statistics are published for the next five years, owing to the disinclination of Local Authorities to enforce measures in whose effect they had no confidence, we know the disease continued to prevail extensively. At this time the pinch of agricultural depression came to be more keenly felt, and the losses from foot-and-mouth disease to be realised. It was necessary to succour all resources and to check every possible drain on them. With this and the growing intelligent interest of the stockowner and landowner came that willing co-operation, which was, and is, essential to the suppression and exclusion of such a highly contagious disease as foot-and-mouth. Accurate returns were now made, and it was realised that in those periods in which the disease became widespread in Great Britain and Ireland many animals were found affected when landed at our great import wharfs. A rising trade in live stock and dead meat with America, Australia, etc., where foot-and-mouth disease had no permanent existence, made us less dependent for our food supply on the Continent. International intercourse between the Animal Departments of States became more intimate, and, consequently, our knowledge of the existence of the disease abroad more complete and dependable. The keynote was struck with the prohibition to land animals from countries in which foot-and-mouth disease was known to exist, the legislative measures compelling Local Authorities to act,

and the settling of limits of infected areas and other actions by the Central Authority.

In 1886 only one outbreak, involving 10 cattle, was recorded. In 1887, 1888, 1889, 1890, 1891, no cases were reported. In 1892, after an entire absence for five years, the disease was reported for fifteen counties, and there were ninety-five fresh outbreaks, with 1248 cattle, 3412 sheep, and 107 swine affected. In 1893, it existed in two counties, there being two fresh outbreaks, and 30 cattle affected. In 1894, we find three fresh outbreaks in three counties, and 7 cattle and 261 sheep affected. For 1895, 1896, 1897, 1898, 1899, there were no returns. In 1899 and 1900, foot-and-mouth disease was very prevalent in France and Holland. In 1900, Essex and Wilts were the seats of small outbreaks. In the present year, Kent and Suffolk are at present enjoying attention. It has been calculated that in Great Britain (exclusive of Ireland)

between 1839-1879, 6,000,000 animals were affected

" 1880-1885, 500,000 " " "

" 1892-1893, 5,300 " " "

Without attempting to deal exhaustively with any phasis of this disease, I think it may not be regarded as out of place if we consider some points concerning its nature and symptoms which have a practical bearing.

Foot-and-mouth disease, or eczema epizootica, epizootic aphtha, vesicular aphtha, murrain, aphthous fever, is acutely contagious, being communicated by contact of diseased with the healthy and by transmission of its contagion by different media. No disease with which I am acquainted so rapidly assumes the epizootic form, or so soon spreads over extensive areas.

It is believed by some that this was originally a disease of cattle; we know that cattle, sheep, and pigs are the most common subjects of its attack. Goats, wild ruminants, dogs, cats, poultry, and man, are said to be sometimes affected. Without desiring to throw any doubt on the possibility of any of these animals acquiring the disease, we may take it that it is only under extraordinary circumstances that this happens. With a somewhat extensive personal acquaintance with the disease, I can state that I have never observed a naturally contracted case in other species than the ox, sheep, and pig.

Ordinarily foot-and-mouth disease is not fatal. The rate of mortality, however, varies considerably. Thus, for six weeks, ending 3rd August 1883, of 30,985 animals attacked, 82, or $2\frac{1}{2}$ per 1000, died. For five weeks, ending 29th September 1883, of 129,036 animals attacked, 1075, or $8\frac{1}{2}$ per 1000, died.

Circumstances, however, occasionally render the percentage of deaths much higher. It would appear that some outbreaks have been much less benign than others, and this has been attributed to variation in potency of the virus and of the vulnerability of the attacked, while it is more than probable that at times complications due to filthy surroundings and contamination with other pathogenic organisms have been responsible, while some forms of treatment, such as poulticing, etc., were certainly followed by disastrous losses. Long road journeys, voyages by sea, and other conditions which debilitate the subject or aggravate the lesions increase the proportion of fatalities, and when

affecting animals on board ship, with rough weather, the losses have been immense. On pregnant animals the effect is rendered serious by a great tendency to induce abortion. It is in the young that the effect is most severe, and foot-and-mouth disease amongst calves, lambs, and young pigs sometimes claims 50 per cent. of deaths.

Virus.—Though of the contagious nature of foot-and-mouth disease there is no room for question, I am not aware that the form of the virus has yet been demonstrated. Many scientists have placed on record their discovery of the actual causal agent, and described its physical characters. Klein, Nosotti, Schottelius, Kurth, Starcovici, Furtuna, Strutzer, Siegel, Bassenius, and others have each described bacteria as the pathogenic factors, while Piana, Fiorentini, Behla, and Jürgens claim to have found certain minute protoplasmic bodies with active amœboid movements which they regard as the virus of the disease.

Experiments of the Berlin Commission of 1897, etc., recorded by Professors Drs Loeffler and Frosch, prove that neither of the above is the cause of foot-and-mouth disease. The discovery and demonstration of the actual germ is perhaps an important matter. It has much concerned our Continental neighbours, some of whom have offered high premiums to the discoverer, but the nearest point that has yet been reached appears to be that recorded in the Report of the Berlin Commission. It was by them found that, if the contents of a fresh vesicle mixed well with water were passed through a filter, the filtered lymph in which no bacteria were discoverable was quite capable of setting up the disease, thus indicating that the virus had passed through the filter. When, however, the lymph was passed through a Kitasato filter, it would no longer infect susceptible animals. The inference is that the undemonstrated virus is a very minute corpuscular element which passes through an ordinary filter but will not pass through the finer one. It is probably too small for recognition by the most powerful microscope now available. Beyond this we know nothing of the physical characters of the virus of foot-and-mouth disease. A much more important matter, from a practical point of view, is the period during which the virus remains effective outside the body of an affected animal. It may, I think, be taken for granted that the virus is an obligatory parasite, and that it does not grow and multiply under natural conditions outside the body of a susceptible animal.

Virus.—Common observation appears to indicate that under ordinary circumstances the virus of foot-and-mouth disease does not retain its effectiveness long. It is unfortunate that our knowledge on this point is not very definite. The longest period which I have heard suggested is referred to in the Annual Report of the Board of Agriculture for 1899,¹ where it is stated that the expert advisers of the French Government hold the view that if the sheds in which diseased animals have been kept are not properly cleansed and disinfected, the specific organism may retain its virulence for several months. Some laboratory experiments would indicate a much shorter period of life. The Berlin Commission found that fresh lymph from a vesicle in a capillary tube was infective to the calf and pig after fourteen days. After three weeks less active; but many samples kept



in an ice-chest for eight or nine weeks were capable of setting up the disease when injected in large quantities.

24 hours drying at	87·8 F.	rendered lymph ineffective.
12 „ heating at	96·6 F.	„ „ „
$\frac{1}{2}$ an hour „	158·0 F.	„ „ „
$\frac{1}{4}$ „ „	140·0 F.	left lymph infective.

These experiments cannot be regarded as exhaustive ; they do not touch conditions which ordinarily exist in nature. At any rate, in an ice-house the virus remains effective for nine or even sixteen weeks ; and it may happen that, between the temperatures of summer heat and freezing point, temperature and other circumstances may favour the vitality of the virus. These laboratory experiments appear to show that low temperatures are more favourable than high, also that drying is very destructive. That hay, straw, etc. on which the slaver has been deposited remains capable of infecting animals for four days I have myself proved by experiment (Derby, 1884).

The virus is contained in the lymph of the vesicles, and, when these rupture, in the saliva, in the milk (Berlin Commission), in the blood up to the time of formation of vesicles, and (according to some writers) in expired air, all the excretions, tears, urine, fæces, nasal and vaginal mucus. I know of no proof of these latter assertions.

An animal is believed to lose the power of transmitting the disease soon after healing of the lesions, though, on account of the possibility of animals carrying the virus obtained from other affected animals with which they have been in contact, it would be rather dangerous to act on this assumption.

In considering the means by which foot-and-mouth disease is introduced and transmitted, I think it will be agreed that the living affected animal is a factor of great importance. The movement of affected cattle, sheep, and pigs, or of any of these, from a situation in which the disease exists or has existed, is a particular cause of spread. In Great Britain, at the present time, the law prohibits the landing of animals from any country in which the disease is known to exist, and, according to official returns, since 1889 no single case of foot-and-mouth disease has been found in any foreign animal landed on our shores. This fact certainly speaks volumes for the methods adopted by our Central Authority. Notwithstanding these regulations and this freedom from the disease at the lairs in which foreign animals landed for slaughter are kept, in 1892, 1893, 1894, and 1900, and now in 1901, the disease has appeared in England. It is of course difficult to escape the conviction that the virus has been introduced by some media other than affected animals. Undoubtedly the virus may be so transmitted, and there must always remain a degree of danger when foot-and-mouth disease is existent in France and Holland and other places with which we are in communication by travellers, importation of hay, straw, etc., whether this be for fodder or as packing. The late Prof. Duguid informed me of his conviction that the disease was once introduced by straw in which eggs were packed. At other times drovers passing over from the Continent may be carriers of the contagion. Indeed, both as to the importation of the virus and the spread after imported, the greatest

regard must be had for mediate contagion. Outbreaks are often explainable only on these grounds. Even accepting this view as to the spread of the disease, it is often impossible to discover the connection of fresh outbreaks with pre-existing cases. For instance, in 1900 there were a few cases in Essex, then suddenly some animals in Wiltshire were attacked, and in course of a few weeks the disease made its appearance in Warwickshire or Staffordshire, and again as unexpectedly at Harlow, on the Essex and Hertfordshire border, while the present century was young when it was reported in three centres in Suffolk, and now we hear of it in Kent. It is also worthy of remark that so many outbreaks have first occurred in the eastern counties in most direct communication with the Continent.

Foot-and-mouth not being a fatal disease, and now one with which the rising generation of stock-owners and possibly veterinary surgeons and even policemen are not familiar, it is possible that some time occasionally elapses before the disease is recognised and reported, while equally possibly some cases may have altogether escaped the notice of the authorities. Indeed, in some instances it may never have been detected or distinguished in the first instance.

Transmission.—Experiments show, though there is some variety in this respect, that usually only very minute quantities of lymph are required to set up the disease.

Susceptibility.—Cattle are much more readily affected by artificial means, pigs less readily, and sometimes sheep with difficulty. In many cases attempts to infect rabbits, guinea-pigs, dogs, cats, rats, house mice, field mice, hens, and pigeons,¹ by inoculating fresh lymph into the mucous membrane of the mouth, into the peritoneal cavity, the limbs, and feeding, did not prove successful. It is not intended to suggest that all these animals are immune, but the experiments indicate that artificial infection by these methods is not always successful.

In all animals subcutaneous injection of lymph usually fails. Injection of the virus into the blood stream and rubbing it into the scarified buccal membrane and skin at the coronets is most often effective. If virus be injected into the blood stream, lesions appear simultaneously in feet and mouth, and sometimes, but more rarely, at the udder. The same effect follows rubbing of the virus into the abraded buccal mucous membrane.

It is not certain whether the virus enters the unbroken skin; probably not. The same may be said as to entrance in inspired air by the air passages. The more common point of entrance is believed to be the alimentary tract, while we know that slight abrasions very often exist on the skin at the coronet, and these afford easy entrance to the virus.

The Incubation Period is very short. If lymph is injected into the blood stream, vesicles commence to form in the mouth, sometimes in the udder, in from twenty-four to forty-eight hours, and soon after about the coronet. With this, as with other contagious diseases, the incubative stage will to some extent depend on the quantity and quality of the virus. When applied to the buccal mucous membrane the temperature rises in from thirty to sixty hours, and in from three to five days vesicles make their appearance.

¹ Berlin Commission, 1898.

Duration.—Foot-and-mouth disease runs its course, if uncomplicated, in from nine to fourteen days. In an outbreak from 30 to 80 per cent. of cattle become affected—*i.e.*, if they are grazing or herded together. This will, of course, largely depend on circumstances.

Immunity.—Naturally a small proportion of animals (cattle, sheep, and pigs) enjoy a very considerable immunity or resistance, while the vast majority are highly susceptible. The period during which animals are immune after an attack varies materially; probably it averages no more than six months. It has been asserted that a cow has had three attacks in one year. This assertion I regard with much suspicion.

Immunity can be artificially induced with a mixture of blood and lymph from a vesicle. The consideration of this matter has less interest for us than for our Continental neighbours,¹ who are not so favoured by insulation.

Diagnosis.—That aspect of the subject which under present conditions most concerns the veterinary surgeon is diagnosis. As a matter of fact, in his capacity as a private practitioner all he is called on to do is to advise his client whether his animals are or are not affected with foot-and-mouth disease, or with anything which renders him suspicious of foot-and-mouth disease. In some of its phases, and particularly where only a single animal is affected, the inexperienced may find some difficulty in arriving at a conclusion. Comparatively recent circumstances, too, show us that it is possible for men of large experience to err. There are, however, certain characteristics associated with the disease which, if carefully regarded, seem to us safe to rely on. Here I propose to speak of the symptoms in cattle, sheep, and pigs.

Though the virus from animals of one species is capable of inducing the disease in animals of the other species, there are some differences in the clinical features which render it desirable they should be referred to separately. At the outset we may remark that whereas it is the rule in cattle to find co-existent lesions in the mouth and feet, such occurrence is less common in sheep and pigs, in which the lesions are often confined to the feet. I have found under special conditions, such as obtained among animals closely packed on board ship or where affected animals have been driven long journeys and then crowded into a yard, that among affected cattle the incidence and extent of lesions have been greater in the feet, but I never saw an outbreak among cattle in which a considerable proportion had not mouth lesions also. I never saw an outbreak in sheep or swine in which the foot lesions did not predominate, and if I were informed that a flock of sheep or herd of swine had a disease confined to the mouth, the feet being healthy, I should have difficulty in being persuaded that, if correctly described, such disease was foot-and-mouth. These points would appear to deserve greater attention than has been sometimes given.

In cattle the first observable abnormality is acute fever. Two three, or four days after infection the temperature is markedly raised sometimes reaching 106° or over. With the appearance of vesicles the temperature falls, and after their rupture it soon becomes near the normal.

¹ Berlin Commission, *ante*, *loc. cit.*

Naturally, in the ordinary course of events, the veterinary surgeon is not called to the first case until this initial stage is past, but if animals have been congregated he will in all probability find plenty of subjects for thermometrical observation. If the animals are at pasture they will usually be isolated and disinclined to move. The common symptoms of fever, scanty fæces, high-coloured urine, decrease of milk, dry hard coat, tucked-up abdomen, and general *malaise* will be present. The appetite is usually interfered with. The other clinical features are associated with the lesions, and probably, notwithstanding the existence of the foregoing, lameness or smacking of the lips will be the first thing to attract attention. Indoors the animal usually lies down, and is made to rise with difficulty. As a result of the irritation connected with formation of the vesicles on the feet, cattle at grass and indoors move the feet restlessly, and often kick as though trying to throw off some irritating foreign body, the rattling sound made with the claws against each other being very suggestive to the experienced practitioner. Now the skin about the coronets is hot, tender to the touch, and somewhat swollen. Soon, at some part of the skin, described as between hair and hoof, the vesicles appear, at first as hard bodies about the size of a pea, gradually increasing in size, remaining discrete and about the size of half a pea pod, or coalescing so as to occupy sometimes the whole of the skin between the hair and hoof. The vesicles are usually greyish-white in colour. Depending on various circumstances, as virulence of the poison, thickness of the epithelium and mechanical injuries, in course of one, two, or three days the vesicles rupture, exposing bright red bases, really the hyperæmic corium, and more or less ragged edges of epithelium. The lesion manifests no tendency to ulceration; the base first becomes covered with an albuminous material, and then by epithelium from the sides. It is, however, easy to understand that, exposed to filth and injury as they are, complications are liable to occur, and it not infrequently happens that suppuration takes place, and the claws are separated and drop off. One, two, three, or all four feet may be affected.

The effect of the virus on the mouth is first noticed as a more or less diffuse redness of the buccal mucous membrane. On passing the hand over the inside of the lips, point or sides of the tongue, little hard bodies like millet seeds are felt. These rapidly enlarge to form greyish-white vesicles, varying in size from that of a sixpence up to that of the whole tip of the tongue. In course of two or three days the vesicles rupture, leaving a typical lesion, a bright red base and edges of more or less ragged epithelium. Healing by ingrowth of epithelium is rapid, and usually complete within fourteen days. Occasionally lesions are found on the borders and outside of the lips, more frequently on the muzzle, whose whole surface is often covered with accumulated epithelium, which, on being roughly rubbed, becomes detached, leaving a bright raw surface. In my own experience the vesicles have been much less commonly observed on the inside of the lower lip than on the inside of the upper lip. On the lesions, which occasionally occur on the outside of the lips, there may sometimes be a slight scab, but an extensive scab is not characteristic.

Such is the picture on examination of the mouth, in which the lesions usually appear a little in advance of those in the feet. There

are, however, connected with the oral changes very attractive symptoms. In the earliest stage the mouth is sometimes kept firmly closed, but soon the irritation excites the flow of saliva and induces the animal to smack its lips and slaver. A ropy saliva, often mixed with shreds of epithelium, drops or hangs from the mouth. The conjunctival discharge is sometimes profuse, running over the face or accumulating in the inner canthus. Occasionally similar lesions are found on the teats and udder, and, according to some writers, elsewhere on the skin, but these are not typical.

In the sheep the vesicular lesions are in the majority of cases confined to the feet; their situation, appearance, and changes are similar to those of cattle. Under some conditions the claws separate and drop off, and when this happens the separation is from above to below, and the shed claw shows no sign of disease in itself. Foot-and-mouth disease does not disintegrate the horn at the solar surface or elsewhere.

When lesions appear in the mouth of the sheep they are usually relatively smaller than those of cattle, but of the same nature. The skin on the outside of the lips and face is rarely affected. It is said that vesicles are occasionally observed inside thighs, arms, and about the vulva. Among the other general effects of fever in the sheep, hurried breathing is often very attractive.

Foot-and-mouth disease in the pig is usually manifested by lesions in the feet and the consequent symptoms. The mouth and snout are occasionally the seats of vesicles. It is believed that pigs are often infected through drinking milk, and so, in an outbreak, become affected after cows. It is necessary that too much importance is not attached to this common order of things, for it has happened, and may again happen, that pigs have proved to be the originators of outbreaks. The filth with which swine are usually in contact renders complication of the foot lesion common.

In pregnant animals foot-and-mouth disease often induces abortion.

In very young animals of all species local lesions are rarely seen. Sucklings of affected mothers often succumb rapidly without manifesting any distinctive symptoms; sometimes dying as though of apoplexy, and, with the exception of some evidence of congestion of the vessels of the alimentary tract, showing no abnormalities on *post-mortem* examination.

As to the differential diagnosis, I venture to think that if all the aforementioned circumstances be taken into careful consideration little difficulty is likely to be experienced. In case of cattle there is no recognised contagious malady in which fever is followed by vesicles in the mouth and feet. A condition of the feet, which on superficial consideration might give rise to some suspicion, is that resulting from cattle walking through some irritant, such as unslaked lime. The mouth is unaffected, and the lesions confined to the lower parts of the limbs do not present the characteristic vesicles, but give evidence of the caustic agent. Lime may be observed on the feet and legs, and there is no suggestion of contagion.

Blain occurs in isolated cases, the feet are not affected, the rapid swelling often involving the skin of the face, and its appearance and disappearance are sudden.

Though sheep are liable to other affections of the feet and limbs,

the seat and nature of the lesions, and the evidences of contagion of foot-and-mouth are, I venture to think, sufficiently distinctive. I have elsewhere referred to the evident distinction between this affection and foot-rot, with which we are all intimately acquainted.

Recent events tell us that it is important to bear in mind that sheep are not uncommonly the subject of a disease of the mouth, lips, etc., known as aphtha. First, it is essential to remember that foot-and-mouth disease is rarely, or *never*, confined to the mouth of sheep, while the tendency of the dead scabbing material to remain on the lips in aphtha creates a picture quite unlike that of the vesicles of foot-and-mouth disease.

Treatment.—In the treatment of affected animals the veterinary surgeon, as a private practitioner, has no discretional function. As an inspector of the Local Authority, or of the Board of Agriculture, his duties are clearly defined by acts and orders.

Prevention.—The greatest importance attaches to prevention, and the most valuable assistance the veterinary surgeon can lend is to know the disease when he meets it, and to use all means at his disposal for setting in motion the machinery of the law. If he diagnoses foot-and-mouth disease, or anything which causes him to suspect it, he should immediately acquaint the owner with the fact, at the same time informing him of his duty, as to disinfection, restriction of movement, and reporting to the nearest police officer, who communicates by telegram with the Board of Agriculture.

As matters are at present, and have been for some years, the procedure adopted is that of stamping out by slaughter of affected and in-contact animals, restriction of movement, and disinfection. These may be carried out under direction of a veterinary inspector of the Board, an inspector of the Local Authority acting for the Board, or by a travelling inspector.

Favoured as we are by our insular position, our main attention must be directed to preventing admission from foreign countries. Our laws as at present seem sufficient as regards importation of live animals. It should be made a penal offence for any drover to pass from a foreign part, in which foot-and-mouth disease prevails, without evidence of disinfection, and also for anyone to import any fodder, or other article, liable to contamination from a similar source. The law relating to the disease in this country is so clear on the former point that I need not refer to the media which are most enumerated. I will again say that, as far as we are concerned, the early suppression and extinction will depend on the early recognition of its existence, and immediate action of the authorities. This requires the co-operation of the stockowner.

It must be borne in mind that one affected animal brought into any of our large markets may kindle the flame. Happily, however, the stockowners of the country are alive to the danger, and have confidence in the action of the Central Authority.

Lastly, I cannot forbear expressing the satisfaction with which we must regard the work of the veterinary profession in its connection with the extinction and exclusion of foot-and-mouth disease. This has not been accomplished without the co-operation of the stockowner, whose sympathy it was, perhaps, a little hard to enlist, but I think it would be difficult for any Government Department to point

to more complete and useful work for the State. What has been accomplished in the name of the Department has been accomplished by the exercise of the professional functions of veterinary surgeons all over the country. We may modestly, yet fearlessly, point to the present freedom from foot-and-mouth disease, pleuro-pneumonia, cattle-plague, and variola, as evidence of our usefulness to the State, and I take it that on no better basis can we rest our claim as a profession for the respect of the community of which we are part.

Official Regulations.—The more important regulations in force for the prevention of foot-and-mouth disease may be summarised as follows:—

Every person having or having had in his possession or under his charge any animal affected with or suspected of foot-and-mouth disease, shall with all practical speed give notice of the fact to a constable of the police force for the police area wherein the animal so infected or suspected is or was.

The constable shall immediately transmit the information by telegraph to the Secretary of the Board of Agriculture.

The constable shall also forthwith give information of the receipt by him of the notice to an inspector of the Local Authority, who shall forthwith report the same to the Local Authority.

This inspector of the Local Authority shall then with all speed proceed to the place where such disease exists or suspected disease is supposed to exist, and discharge duties and powers imposed and conferred on him as inspector by or under the Act of 1894 or the Order.

The inspector shall report forthwith to the Secretary of the Board of Agriculture.

Every inspector on leaving premises wherein foot-and-mouth disease exists or is suspected to exist shall wash his hands in soap and water and disinfect his boots and clothes.

The Local Authority must consult a veterinary inspector or a veterinary surgeon.

Animals shall not be moved into or out of an infected place except with a licence of an inspector or officer of the Board.

Carcases are not to be removed except with permission in writing from an inspector or officer of the Board of Agriculture.

Fodder, litter, dung, utensils, pens, hurdles, etc., may not be removed except after thorough disinfection and with permission.

No person, except the person tending the animal, shall enter any shed, field, or other place, being part of an infected area, except by permission, in writing, of the inspector.

Every person leaving any such shed, field, or other place shall thoroughly disinfect himself.

Any person tending a diseased or suspected animal shall not tend any animal not so diseased or suspected without permission in writing.

Dung, fodder, litter, or anything that has been in an infected place, etc., shall not be sent or carried on a railway, canal, river, or inland navigation, coasting vessel, or a highway or thoroughfare, except with a licence of an inspector or officer of the Board of Agriculture, or of an inspector of the Local Authority.

An inspector may give notice in writing to the owner or person in

charge prohibiting movement of any animal from out of any pen, field, shed, sty, or other place. An inspector may withdraw such notice.

The inspector shall with all practicable speed send copies of any and every notice to the Local Authority and the police officer in charge of the nearest police station of the district.

Carcases of animals which have died of foot-and-mouth disease shall be buried in the skin (which must be slashed), six feet deep, and covered with quicklime, or after being thoroughly disinfected may, with authority of the Local Authority or licence of the Board, be removed to a slaughter house for destruction by fire or by chemical agency.

Digging up of a carcase is unlawful, except by licence of Board or written permission of an inspector of the Board.

Any shed or other place in which an animal affected with foot-and-mouth disease has been kept shall be swept out, and all litter, dung, and other things which have been in contact with or used about any such animal shall be removed therefrom. All possibly contaminated places shall be washed, scrubbed, or scoured, and sheds limewashed.

Litter, dung, etc., shall be disinfected, burnt, or destroyed. According to the regulations of the Local Authority, this disinfection shall be carried out by the occupier of such shed at his expense, or at that of the Local Authority. If the Local Authority has made no regulation on this point, all expense shall be borne by the Local Authority. If the owner will not disinfect, the Local Authority may step in and do it, and to recover all expense from such occupier.

The Local Authority may make regulations for removal within their district.

The Local Authority may make regulations as to markets, sales, etc., and may seize animals affected with foot-and-mouth or those in contact or which have been in contact. Such animals may be detained in a convenient place, but they must be isolated, and can only be moved with sanction of the authorities.

The declaration of infected area is made by the Board of Agriculture *only*.

The inspector of the Local Authority shall report to the Local Authority all proceedings taken by him, and the Local Authority shall forthwith report to the Board of Agriculture.

It is unlawful to expose a diseased or affected animal in a market, fair, saleyard, or other public or private place where animals are commonly exposed for sale, or to place it in a lair accessible to other animals.

The Local Authority may, if they think fit, cause to be slaughtered any cattle, sheep, or swine affected or suspected of being affected, any such animals having been in contact with the affected or exposed to infection.

The Local Authority shall, out of the local rates, pay compensation as follows for animals slaughtered under such article :—

When the animal slaughtered was affected with foot-and-mouth disease, the compensation shall be the value of the animal immediately before it became so affected ; and

In every other case the compensation shall be the value of the animal immediately before it was slaughtered.

The Board of Agriculture reserves the right to give permission for movement of any animal, carcase, or thing when they may think exceptional circumstances render the movement necessary or expedient.

Any powers conferred by the Orders on a Local Authority or an inspector of a Local Authority may at any time be exercised by the Board or an inspector of the Board.

The provisions of the Foot-and-Mouth Disease Order, except where it is otherwise provided, shall be executed and enforced by the Local Authority.

When an inspector of a Local Authority finds that foot-and-mouth disease exists, or has within ten days existed, in his district, he shall forthwith make a return thereof to the Local Authority and to the Board, and shall continue to make a return thereof every Saturday until the disease has ceased.

VETERINARY DAIRY INSPECTION.

By JOHN MALCOLM, F.R.C.V.S., Birmingham.

The regular veterinary inspection of dairy cows kept for the public sale of milk has been advocated for many years by veterinarians taking a special interest in questions of public health. These veterinary sanitarians clearly recognised the dangers of infected milk, and did their best to awaken public opinion on the question. For long apparently making little headway, they nevertheless paved the way for the general and systematic veterinary inspection of dairy cows at present in process of realisation. At the same time these pioneer advocates will readily concede that the first real step forward in the movement came not directly from them, but from the Royal Commission on Tuberculosis. This commission's report contained clear experimental evidence of the dangerous character of the milk of cows affected with tuberculosis of the udder, and uncompromisingly condemned its use. As a natural sequel to this report the Local Government Board issued the Dairies, Milkshops, and Cowsheds Amending Order, 1899, which gave Local Authorities power to deal with tuberculosis of the udder in dairy cows on the same footing as other infectious diseases scheduled under the 1885 order.

It is at once clear to anyone reading this new order that it could not be enforced except through a qualified veterinary surgeon; and as the chief Urban Authorities in the interests of public health resolved to put the provisions of the order immediately in force, they naturally proceeded to appoint veterinary dairy inspectors, and thereby at once established the veterinary inspections of dairy cows on a firm footing. To the many Authorities who have already taken action in the matter others will no doubt continue to be added, until in all probability the great majority of the municipalities have enforced the new regulations.

But the laudable desire of Urban Authorities to secure the benefits of this order has not, so far as my information goes, awakened any kindred desire on the part of Rural Authorities. On the contrary, they appear to ignore its existence, and to allow its regulations to remain a dead letter.

Why this difference in the procedure of Town Councils and County Councils is a question of some interest. The fact that towns people are mostly buyers of milk, and many country people sellers of milk, can scarcely have any bearing on the point. The difference probably is connected with a greater interest by the former in questions of local government, and a greater disinclination by the latter to enforce innovations of any kind.

Be that as it may, the difference unquestionably exists, and its effect is to materially diminish the value of the order. An urban district may secure that the milk produced within its boundaries is obtained from healthy cows, but as a rule it has no such control over the milk obtained from outside, and as the major portion of the milk supply is usually obtained from the outside rural districts where Authorities neglect to enforce the Dairies Order, it follows that the milk of cows affected with tuberculosis of the udder, if kept in outside districts, may be publicly sold in a district with perfect impunity, and in spite of every care on the part of the Local Authority. Not only does the order not provide for uniformity of action by contiguous Authorities, but it omits to say what is to be done with condemned cows. These points intelligent dairymen fully appreciate. The other day I condemned a cow with tuberculosis of the udder, and ordered her removal from the dairy stock. The owner has two farms, one inside, another outside our boundaries. In reply, "I have been considering," said he, "what you would do if I took her to my other farm. You have no power there, and the County Authorities never visit me." This dairyman was too honest to take any advantage of these defects in the order, and he had his cow killed, but another dairyman had no such scruples. After a cow belonging to the latter had been condemned he surreptitiously removed her at night, and some considerable time after admitted that he had sent her to be sold at a neighbouring public auction.

The recognition of these and other defects has induced a number of Municipal Authorities to obtain extra powers by special Act of Parliament, enabling them to follow suspected milk to its source, and there examine the suspected cows. The conditions attached to these special Acts, however, seriously limit the benefits they are intended to confer. Where such an Act has been obtained the Local Authority, before putting it in force, must have reasonable grounds for suspecting that a particular milk contains the produce of one or more cows affected with tuberculosis of the udder, and as the Authorities must chiefly depend upon the microscopic examination of the milk for this evidence, since it is at least unlikely that powers will be generally granted to experiment on guinea-pigs for diagnostic purposes, there must necessarily follow no inconsiderable augmentation in the number of municipal dairy microscopists if these Acts are to be of real use. Having obtained sufficient data for suspecting any particular supply, the next step is to obtain an order signed by two local magistrates authorising the veterinary inspector to examine the cows of the incriminated dairy. Then, after due notice given to the dairyman, the official inspection of the cows may take place, and in the event of any cow being found with tuberculosis of the udder the sale of her milk is interdicted. Under such conditions it would seem an easy matter for any unscrupulous dairyman to secure the

absence of any suspected cow from his cowshed during this previously notified inspection; and thus there is a clear loophole by which the provisions of these acts may be evaded.

To obtain private Acts of Parliament is usually rather a costly procedure, and they should naturally confer some real benefit; but so long as the private dairy Acts are hedged about with restrictions as at present their utility is somewhat problematical, and in any case they can scarcely prove satisfactory in operation.

If either an order could be obtained securing something like uniformity in dairy inspection under the different Authorities, or a general Act passed giving any Local Authority the necessary power to authorise the inspection of any dairyman's cows whose milk is sold within their jurisdiction, far better results would be obtained than can ever be expected from the present procedure of obtaining private Acts. No dairyman with healthy cows in clean sanitary buildings need fear inspection of his stock, but no one should be free to publicly sell the milk of diseased cows, and the only possible way to prevent this is by a general system of veterinary inspection. Although some apparent defects exist in the present regulations, it must be admitted, after making full allowance for all these, that the new Dairy Order is a great step forward, and has already been productive of incalculable good, not measured in any adequate degree by the number of detected cases of tuberculosis of the udder. Perhaps a few particulars of the result of the veterinary inspection of the dairy cows here may not be uninteresting.

During the last two years the dairy cows within the city have been under regular veterinary inspection. On 31st December 1900 there were on the register sixty cowsheds, registered to contain 608 cows. The veterinary visits were so arranged that the whole of the cows have been inspected periodically, the udder of each cow as a rule being examined once a month. As a result of this inspection last year thirty-six cows were placed under special examination. Of these, eleven had chronic induration of the udder, two had cowpox, twenty were suffering from sporadic mastitis of a suppurative or of a catarrhal character, and two had tuberculosis of the udder. In 1899 thirty-five cows were specially examined. Of these, thirteen had induration of the udder, seven had suppurative mastitis, and twelve had tuberculosis of the udder. In 1900, therefore, there were only two cases of tubercular mastitis or tuberculosis of the udder, against twelve in the preceding year, in a dairy stock averaging 600 cows, but probably covering double the number, as in a general way no cows are kept in the town sheds over twelve months. Last year's favourable results were, I believe, in a large measure due to the extra care exercised by the cowkeepers to purchase healthy cows free from defect or disease of the udder, a rule the majority have habitually observed since the regular and systematic inspection of the cows was undertaken. The cases of ordinary mastitis were more prevalent last year than in 1899, but not more so than appears to be the case in other districts—*e.g.*, in the published record of Mr Simpson's inspection of the London dairy cows, 1·6 per cent. were returned as suffering from mastitis, whereas the twenty-one cases recorded here give an average of under 5 per cent. at each inspection.

In the majority of these cases the disease only affected one quarter

of the udder. In some of the more severe cases either the cowkeeper or the milkman had recognised the altered condition, and the sale of the milk was discontinued; but in the majority of the mild cases the condition had not been noticed, and the milk was being mixed and sold with the other milk in the usual way until its altered character was pointed out and its sale stopped. As a rule the mild cases of simple catarrh recovered, but in the more severe cases the secreting function of the quarter was lost. With respect to the two cases of cowpox, the disease existed in its incubative stage when the cows were purchased, and as the result of the preventive measures adopted the disease did not spread.

Considered comparatively, the dairy cows in the city are of a good type, and, generally speaking, have sound udders.

The duties of a veterinary dairy inspector are materially facilitated and rendered much more pleasant when he possesses the confidence and the respect of the cowkeepers. The veterinarian must not only be impartial but considerate, as well as just, in all his procedure; he should be observant, promptly noticing visible abnormal conditions, and his examination should be thorough, thereby readily detecting the less apparent changes whose existence are generally known to the dairymen.

In casually walking behind the cows in a cowshed any marked departure from the normal in the size or shape of the udder can readily be recognised, but may be of no consequence from a public health point of view, as in the case of a non-secreting atrophied quarter, which anyone can see. On the other hand, serious morbid changes may exist, gravely altering the character of the milk, without any apparent visible alteration in the udder, as in some cases of parenchymatous mastitis and in the early stages of tuberculosis, which require both a careful manipulation of the gland and a thorough examination of its secretion for their recognition.

The differential diagnosis of tuberculosis of the udder has been very fully and clearly dealt with in a recent number of this Journal by the editor. At the same time the subject is such an important one that no harm can be done by shortly recapitulating the chief points to be relied upon for its diagnosis in practical inspection.

In tuberculosis of the udder one or more quarters may be affected, a posterior one being most frequently involved, although by no means invariably so. The disease usually commences as a slow indolent swelling in the depth of the gland, thence gradually extending, increasing in size and density until the whole quarter, and in some cases even the greater part of the organ, is invaded and markedly enlarged. This extension may take place in six or eight weeks, but it may be much longer. The change is distinctly a chronic one, there being little or no pain, heat, or other sign of an acute character accompanying it. The enlarged udder is somewhat hard and fleshy to the feel, and there is enlargement of the inguinal lymphatics, which, however, may be difficult to recognise in cows having naturally large fleshy quarters.

In the other forms of mastitis, except that due to actinomycosis, after the acute phenomena have passed, atrophy of the gland usually sets in, but, as already indicated, the contrary is the case in tubercular

mastitis, there here being invariably a permanent increase in the size and density of the organ.

When first examining such a case the milkman generally confidently asserts there is nothing the matter with the cow's udder, nor ever has been, and, further, assures you the milk is all right, in proof of which he at once milks the suspected quarter and asks you to inspect the milk. He little realises that in a case of indolent gradually extending induration of the udder the fact that the milk retains its normal appearance for a time is a very grave symptom, and, to say the least, very highly suspicious of the existence of tuberculosis of the udder. In the latter stage the secretion becomes modified, lessened in quantity, and transformed into a more or less watery liquid.

As a valuable aid to diagnosis the tuberculin test may be utilised. It at least establishes the existence or non-existence of tuberculosis in the patient, and it may assist in differentiating between tuberculous and actinomycotic mastitis. It, however, conveys no definite information as to the actual condition of the udder, and cannot, therefore, be assigned the position of more than a corroborative factor in diagnosing disease of that organ.

It is clear, therefore, that although tuberculosis of the udder can be diagnosed by an expert inspector with almost unerring certainty, still the positive diagnosis must depend upon the presence and recognition of the causal organisms. In this connection extirpation of the inguinal gland or extraction of a portion of the diseased udder by means of a harpoon for microscopic demonstration has been suggested, but I do not know any inspector who practises this. There remains, therefore, the bacteriological examination of the milk. It is generally conceded that the most reliable results can be obtained by the intraperitoneal injection into guinea-pigs of infected milk, but this is only open to a very limited practice. In general practice one has to depend upon the microscopic detection of the bacilli in the sediment or cream of infected milk.

Subacute catarrhal mastitis first appears as a gradual hardening at the base of the teat and around the galactophorous sinus. There is a very slight degree of pain, no marked enlargement of the quarter, and no acute symptoms. As a rule the disease is confined to one quarter, and seems to involve chiefly the lining membrane of the sinus and the large ducts. The milk when newly drawn from the gland shows no apparent change, but if allowed to stand until the cream separates it looks watery and almost translucent, while there appears to be a mucoïd material mixed with the cream. On microscopic examination of the milk, epithelial cells, leucocytes, and a few streptococci are recognised. The disease usually runs a subacute or chronic course, and it is some time before recovery takes place and the milk regains its normal character. As a rule some slight thickening and induration remains for a time in the lower portion of the gland. Although no doubt the actual cause of these cases of catarrh is correctly ascribed to bacterial infection, yet the circumstances under which they frequently occur suggest as the existing cause in the origin of some of them some defects in milking. Dairymen at least have no doubt that catarrhal mastitis is less frequent with careful competent milkers than with the careless and incompetent.

Acute parenchymatous mastitis is frequently met with and is easily recognised. It commences as an acute simple catarrh of the glandular mucous membrane, but soon assumes a suppurative character. One or two quarters or even the whole gland may be affected. There is usually well-marked constitutional disturbance, which varies in intensity according to the severity of the attack. Locally there is great pain, heat, and swelling of the quarters affected, frequently with œdema of the neighbouring tissues. The joints, more especially the hocks, are not infrequently swollen. The milk from the commencement is markedly altered in character. In the early stages it has the appearance of a dirty, flaky, serous fluid of a reddish-brown tint. Set aside in a vessel it separates into three layers, precipitated casein at the bottom, a dirty reddish-brown whey in the centre, and a mucoid-like greasy material on the top. Microscopic examination reveals epithelial cells, blood cells, and numerous streptococci. Later the milk becomes thicker, and more or less purulent in character, until, eventually, it appears to be little other than a thick creamy pus.

In very acute cases gangrene and sloughing of a portion or the whole of a quarter may result, but such cases have not been met with here. The usual sequel is the complete loss of the secreting function of the affected quarters, with their subsequent atrophy, but some of the less acute cases seem to be accompanied by interstitial proliferation, resulting in some permanent enlargement and induration of the quarter, in which cases occasionally the milk regains its normal character, but is generally materially diminished in quantity. In other cases, stenosis of the teat duct and accompanying permanent inactivity of the quarter occurs. Sometimes a case is met with in which, although both hind quarters have been affected, yet one may, after the acute symptoms have passed, fail to observe any visible alteration on a casual inspection, but on manipulation, and drawing some of the milk from the teat, the actual condition is at once clearly recognised.

Bacterial infection is now usually assigned as the cause of acute parenchymatous mastitis; at any rate, it has to be admitted that streptococci are present in the lacteal secretion, while they are invariably absent in milk when drawn from healthy udders; and, furthermore, it is noted in practice that the cases rarely occur singly, one case in a shed being usually followed by a number of others.

Subcutaneous and interstitial mastitis is a condition chiefly met with in newly calved cows. The cases usually occur suddenly, and they are frequently ascribed to colds, chills, etc., but, probably, are connected with infection through an injured part of the skin, a process favoured by the intense congestion of the organ which then frequently exists. It is an acute inflammation of the subcutaneous and interstitial connective tissue, usually affecting the half or the whole of the udder, and characterised by heat, pain, and marked swelling. Generally there is little constitutional disturbance, and the milk, though temporarily diminished in quantity, is apparently unaltered. Recovery is the rule, unless the case is neglected or complicated by parenchymatous mastitis.

Traumatic mastitis is most frequently met with in cows with large udders, and is the result of external injuries, the evidence of which is usually apparent. In general the degree and extent of the inflammatory action depends on the severity of the injury, although in some

cases slight wounds may result in an extensive infective inflammation. As a result of traumatic or subcutaneous mastitis small abscesses frequently form in the subcutaneous tissue of the gland. In many cases they do not involve the gland structure or cause any alteration in the character of the milk, though they are a source of considerable pain and discomfort to the animal.

A mastitis of a specific contagious character, apparently quite distinct from the conditions already mentioned, is occasionally met with. In 1898, in Volume XI. of this *Journal*, I recorded an outbreak of a contagious inflammation of the udder amongst some dairy cows in the neighbourhood of Birmingham, and a short summary of the symptoms presented by the animals attacked may not be out of place here. The affected cows showed slight fever and loss of appetite, while, locally, the condition was manifested by a rash on the base of the teats and on the adjacent gland surface, by slight tumefaction and induration of the gland structure surrounding the milk sinuses, by pain on pressure on the indurated regions and during milking, and by a marked alteration in the quantity and quality of the milk, which was much reduced in amount, and when drawn appeared a dirty yellow liquid, containing curdled stringy clots.

On my first visit of inspection, on 16th July, I found three cows suffering from the attack. In each, all the quarters of the udder were affected in varying degree, but there was little or no general enlargement or œdema of the organ as seen in cases of acute mastitis. The disease was in a different state of evolution in each animal. In one it was in the initial stage, in another more advanced, while in the third recovery had commenced. On 27th July Mr Olver of Tamworth inspected the cows with me. By this date one cow had practically recovered, and the other two were progressing favourably, but in the meantime another two cows had become affected. On 28th August I reinspected the cows with Professor Stockman, but by this time three of the number had seemingly quite recovered, and only the two last affected showed any local indication of the disease.

As soon as marked alteration in the milk was observed its sale was discontinued, and it was given to the farm pigs, on which it had no apparent ill effect.

These cases differed from some recorded cases of contagious mastitis by the presence of a well-marked eruption, by a more rapid evolution of the disease, by the absence of a permanent induration node at the base of the milk sinus, and by a final recovery.

The eruptive affections of the cow's udder take an important place among diseases coming under the special cognisance of the veterinary dairy inspector.

Since the Hendon cow disease was accused of giving rise to scarlet fever in the human being, the interest naturally taken in mammary eruptions has been much intensified, but it is of no little significance that although both medical and veterinary sanitarians during the fifteen years since that outbreak have been on the outlook for any eruptive mammary disease in cows capable of transmitting scarlet fever to mankind no testimony of any such causal connection has been forthcoming.

The most important exanthematous disease affecting the cow's

mammary gland is vaccinia or cow-pox. This appears as a vesico-pustular eruption observed almost exclusively on the udder and teats, but one seldom sees it run through its natural course. The pustules usually number two or three on each teat and quarter, although in the more severe attacks there may be four or five. It commences by slight fever and diminution in the lacteal secretion, accompanied locally by redness and some infiltration of the skin and followed by papules and then vesico-pustules, which are, however, almost immediately ruptured by the hands of the milker or by the cow lying down. These vesico-pustules are round or oblong in shape, flattened or umbilicated in the centre, and surrounded by a slightly raised well-defined areola. About the eighth day the vesicles are filled with a limpid fluid, which in the course of another two or three days assumes a thick opaque purulent character. The rupture of the vesicle is followed by the formation of a brown crust of irregular form, under which healing takes place. After the scab has fallen off or been removed a depression or pit remains, and the surrounding skin has a brownish tint. The disease runs through its natural course in about three weeks. If the sores on the teats are much irritated by the hands of the milkers healing is retarded, and the sores may even take on an ulcerous character. A point of much interest in connection with this disease is that not only is it transmitted from cow to cow by the hands of the milkers, but the milkers themselves are often infected from the cows, an eruption appearing on their hands and arms. Similarly the cows may be infected from the eruptions on the hands and arms of the milkers.

Various forms of eruption, known as spurious or false cow-pox, white or blister pox, varicella, etc., are not infrequently met with, and in some respects resemble true cow-pox, but differ from it in some essential particulars. The eruption in these cases is of a vesicular rather than a pustular character, and no distinct papules precede the formation of the vesicles, which are small, superficial, raised on the surface or acuminated, rather than umbilicated, and not surrounded by a tumefied raised areola. The scabs which form are thin, and when cast off do not leave the pit-mark of true cow-pox. The usual duration of false cow-pox is short, the whole evolution taking place in about a week or ten days; but there may be successive crops of vesicles, and the affection may extend over some weeks.

Like true cow-pox, false cow-pox seems capable of transmission from cow to cow by the hands of the milker.

Occasionally mastitis occurs as a complication of the eruption on the teats. It may possibly be connected with fermentation of the milk left in the sinuses in consequence of the difficulty in milking some cows with sore teats. The milk appears as a serous liquid with caseous masses, and later on suppuration is established, in which case the usual termination is loss of the secreting power of the quarter.

A mammary eruption usually occurs in foot-and-mouth disease and occasionally in rinderpest, but in these cases the vesicles are more irregular and variable. Although an areola surrounds them, it has not the tumefied character of that of cow-pox. These eruptions are, moreover, accompanied by the typical symptoms which characterise the affections.

Chaps, cracks, and allied sores on the teats are frequently met with,

but unless neglected are not of much importance from a public health point of view, and do not therefore seriously concern the dairy inspector.

In the interests of economic and efficient administration the veterinary inspector's duties ought to include, in addition to the inspection of cows under the Dairies and Cowsheds, 1899, Amending Order, their inspection under the Contagious Diseases (Animals) Act, as well as the general sanitary inspection of cowsheds, but the consideration of the duties entailed thereby must be deferred until another occasion.

THE RAPID DIAGNOSIS OF RABIES.¹

By MAZŸCK P. RAVENEL, M.D., and J. MCCARTHY, M.D., from the Laboratory of the State Live Stock Sanitary Board of Pennsylvania and the William Pepper Clinical Laboratory (Phœbe A. Hearst Foundation).

A RAPID diagnosis of rabies is a matter of extreme importance for the physician and the patient, as well as for the general public. There is perhaps no other disease which strikes so much terror into the mind of the average person, and the cry of "mad dog" is a signal for general excitement. Any one bitten by a dog supposed to be rabid heretofore has been kept in a state of nervous suspense until it could be definitely determined whether or not the animal was a victim to hydrophobia. Up to the present time the only sure means of diagnosis in a vast majority of cases has been the inoculation of rabbits from a portion of the nervous system of the animal inflicting the bite—a practice often impossible to carry out with any degree of certainty, on account of the poor condition in which much material is collected. Even under the most favourable circumstances, from two to six weeks had to elapse before a positive diagnosis could be made. As well stated by Babes, it would seem "that this disease, so clearly characterised by a train of symptoms, constant in their character, ought also to present characteristic lesions in the nervous centres, and especially in the ganglia which preside over the production of symptoms," an opinion which has been generally accepted and acted upon by many students, their object being not only to disclose the cause of the striking symptoms, but also to find a method of diagnosis more rapid and more certain than any yet known.

The first step in this direction seems to have been due to Pollailon and Nepveu, who described the lesions observed in a man who had died of rabies. They noted that the whole cerebrospinal axis was strongly congested, and that the ganglion of Gasser was hyperæmic and infiltrated with round or oval cells, some of them being hyaline in appearance, and which they considered "probably epithelioid cells from the capsule of the ganglion cells." The work of Balzer, calling attention to the changes in the central nervous system, was followed in 1874 and 1875 by Benedikt's article, both authors describing the distension of vessels in the nervous centres, accompanied by an extravasation of erythrocytes and leucocytes into the perivascular spaces.

¹ Reprinted from the "University Medical Magazine," January 1901.

Kolesnikoff, in 1875, described besides an invasion of the pericellular spaces by round cells occurring in the hemispheres, cerebellum, spinal cord, and the sympathetic and intervertebral ganglia. Schaffer also made a careful study of the vascular and cellular changes found in the case of a man, and called attention to a hyaline and fibrillar degeneration and vacuolation of the cells of the anterior horns of the cord. In 1886 Babes concluded, as a result of numerous researches made in man and in dogs, both natural and experimental cases, that the essential lesion of rabies consisted in an accumulation of embryonic cells in the neighbourhood of the central canal, and especially about the large modified cells of the motor centres of the bulb and cord. Writing again in 1892, Babes reaffirmed his observations and

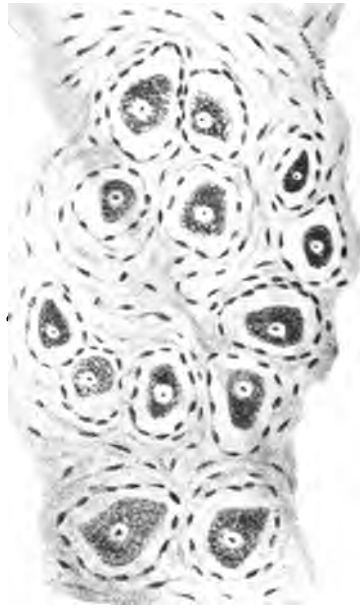


FIG. 1.

Normal ganglion of dog. (Reproduced from *Crocq. Jnl. de Neurologie*, V., No. 13.)

insisted on the profound changes taking place in the nerve cells. He held that it was possible to make a rapid diagnosis of the disease by a microscopic examination of the bulb and cord. He described in the bulb what he considered the diagnostic lesion of the disease—namely, the pericellular accumulations of embryonal cells described by Kolesnikoff, and for which he proposed the name “rabid tubercle.” The cells of the bulbar nuclei undergo degeneration and present the various stages of chromatolysis. There is loss of the prolongations and a progressive modification and even total disappearance of the nuclei, a dilatation of the pericellular space, and an invasion not only of this space, but also of the nerve cells and embryonal cells, and at the same time small corpuscles which are hyaline, brownish, and in parts metachromatic. Many of the nerve cells become surrounded

by a large zone of embryonal cells, and when the cell is completely degenerated these occupy the cell area and constitute the rabic tubercle.

Since 1891 Babes has examined the bulb of 487 dogs, controlling his results always by inoculation of rabbits. His results have led him to trust this means of diagnosis, and he still holds it to be one of the best means of rapid diagnosis at our disposal, although except in Babes' own laboratory the method does not seem to have been practised, at which he expresses a surprise which appears justifiable. Recently Nelis, working with Van Gehuchten, discovered in the spinal ganglia of two men who had died of rabies, and of a number of animals, peculiar changes which they considered to be the diagnostic lesion of the disease. They have confirmed all the lesions described by other authors, but in addition have noted what they consider to

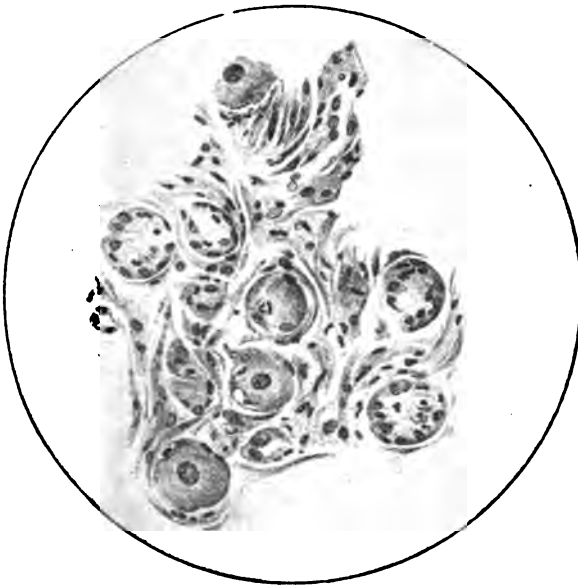


FIG. 2.

Plexiform ganglion of rabbit, dying of rabies produced by subdural inoculation.
The capsules are filled or partially filled with foreign cells.

be more diagnostic than any other. The most profound, the most constant, and the earliest lesions are noted in the peripheral cerebral and sympathetic ganglia, and the changes are specially marked in the intervertebral ganglia and in the plexiform ganglia of the pneumogastric nerve. Normally, these ganglia are composed of a supporting tissue, holding in its meshes the nerve cells, each one of which is enclosed in an endothelial capsule (Fig. 1). The changes characteristic of rabies consist in the atrophy, the invasion, and the destruction of the nerve cells brought about by new-formed cells derived from the capsule, which appear between the cell body and its endothelial capsule. These new-formed cells increase in number, invade the protoplasm of the nerve cell, and finally completely occupy the entire capsule (Fig. 2). These changes are widespread, but few capsules remain unaffected, and in advanced cases the section has very much

the appearance of an alveolar sarcoma. The authors have considered in detail the findings of previous workers, but without exception they consider them as secondary and without any importance, either in the production of symptoms or in making a diagnosis. They hold that Babes has attached an undue importance to his rabic tubercle. In their first publication they held that "these lesions are specific of the rabic infection, if not by their nature, at least by their localisation." In a later note, in answer to objections raised by Nocard and others, Van Gehuchten says that "the lesions of the cerebrospinal and sympathetic ganglia which he and Nelis have discovered are not specific of rabies in general; they are only specific of the disease as it occurs naturally." It is to be noted that the authors have made no claim regarding an early diagnosis of rabies, but only a rapid method. Van Gehuchten says his method "concerns not a precocious diagnosis, but only a rapid diagnosis in rabies." This has been further shown by the experiments of Cuillé and Vallée, who, after inoculating dogs with the virus of rabies, sacrificed them at various times after the appearance of symptoms. They found that in animals in which the disease had just become manifest the lesions in the plexiform ganglion were slight or absent. They conclude that as a means of diagnosis the method of Van Gehuchten and Nelis has great value in the case of animals in which the disease has run its full course, ending in death.

The subject has been studied also by numerous veterinarians of note in Europe, notably Degive and Hébrandt of the State School at Cureghem, and Nocard and Vallée of France, all of whom have obtained results confirming the value of the discovery. Quite recently Crocq has made a most thorough and complete review of the literature on the subject and a study of the method. He believes that Van Gehuchten is mistaken in considering the perivascular and pericellular neo-formations described by Kolesnikoff and Babes as unimportant changes, and believes that by their constant character, their localisation and evolution, they have a considerable diagnostic value. He says: "The lesions described by Babes, and Van Gehuchten and Nelis, are both remarkable. They are so great and so clearly defined that I can understand how the authors, each on his side, may have concluded that the alterations described by them were specific." The procedure of Van Gehuchten and Nelis is, however, more simple and rapid than the method of Babes, and on this account is to be preferred. Our own work leads us to believe that the changes in the intervertebral ganglia are more constant than those in the bulb, as will be seen by reference to the summary below.

The method of procedure recommended by Van Gehuchten and Nelis is as follows: The ganglion is put at once into absolute alcohol, in which it is left for twelve hours, the alcohol being changed once. It is then transferred for one hour to a mixture of absolute alcohol and chloroform; next put for one hour in pure chloroform; then for one hour in a mixture of chloroform and paraffin, and lastly in pure paraffin for one hour. The sections are put in the oven for a few minutes, then passed through xylol, absolute alcohol and 90 per cent. alcohol, after which they are stained for five minutes in methylene-blue, according to Nissl's formula, differentiated in 90 per cent. alcohol, dehydrated in absolute alcohol, and cleared in essence of cajuput and xylol. If frozen sections are cut they are put for a few

minutes in 90 per cent. or 94 per cent. alcohol. In our work we have generally used 10 per cent. formalin for fixing our tissues. They are then transferred to 95 per cent. alcohol, and finally to absolute alcohol. For the most part the tissues have been cut without imbedding, being attached to blocks by the aid of mucilage of gum arabic, though in some cases celloidin has been used. For the bringing out of the chromatolytic changes the Nissl method has proved the best, but the capsular changes were brought out in sections stained by hexatoxylin and eosin. Since these latter changes are the most essential diagnostic features in the sections, we would suggest that material unfit for the Nissl method will still show the capsular changes when stained by hematoxylin and eosin.

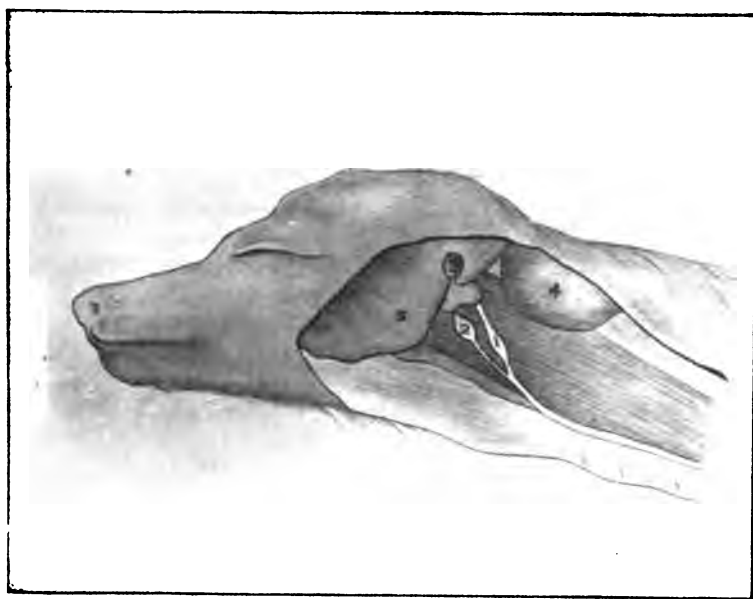


FIG. 3.

Dissection of upper cervical region of dog after Vallée.

1. Plexiform ganglion. 2. Cervical ganglion. 3. External auditory meatus. 4. Atlas.
5. Interior maxilla.

Up to the present time we have examined twenty-eight cases of rabies, including eleven dogs suffering from street rabies, one cow, one horse, and fifteen rabbits which were inoculated from these animals, a few being of the second generation, *i.e.*, inoculated from the first experimental animal. In all of the cases studied (twenty-eight), except that of the horse suspected of rabies, we obtained positive results in the plexiform ganglia. In one case, however, the changes were slight, and more marked in the peripheral (distal) portion of the ganglion. In twenty-one cases the bulb was examined for the rabic tubercle of Babes. Positive results were obtained in nineteen, though in two cases only chromatolysis, without distinct tubercle formation, was present. It will be seen that in two cases the rabic tubercle of

Babes was not found, while the lesions described by Van Gehuchten and Nelis were present, though in one of these the changes in the ganglion were so slight as to leave us in some doubt. In this case, however, the subdural inoculation of rabbits gave positive results, and the microscopic examination of these rabbits showed the bulbar lesions of Babes as well as those of Van Gehuchten and Nelis in the ganglia. In the two cases mentioned in which the bulb showed only chromatolysis without distinct tubercle formation, the lesions in the ganglia were marked. One of these was the cow, which had shown clinical symptoms leading the veterinarian in attendance to suspect rabies. Two rabbits inoculated subdurally with the brain of this cow both died with typical signs of rabies, and by microscopic examination both the rabic tubercle of Babes and the lesions of Van Gehuchten and Nelis were found.

Of the rabbits examined three had been inoculated with virus of the second generation from dogs having street rabies. One of these rabbits showed symptoms of the disease on the eleventh day after inoculation, the shortest time which elapsed in any of our cases between inoculation and the appearance of rabiform symptoms. The microscopic examination of those rabbits gave positive results.

Of the dogs examined six had shown unmistakable clinical signs of rabies, while five were only strongly suspected of the disease. All the cases were controlled by the inoculation of rabbits, except two dogs, in which the clinical symptoms left no doubt as to the diagnosis.

In the summary of our cases we have not included one which occurred in a human being, for the reason that we obtained only a small portion of the upper cord, attached to which was a small fragment of an intervertebral ganglion, not observed until sections were examined under the microscope. In some of the sections a few ganglion cells were found which showed the typical proliferation of the endothelial cells of the capsule. Rabbits inoculated with the cord gave positive results, and some of these have been included.

We take this opportunity of putting this case on record. The patient was a girl eight years of age who came under the care of Dr Frederick Krauss, on 12th August 1900, by whom the diagnosis of rabies was made, and to whom we are indebted for the history. Six weeks before, the child had been bitten on the right ear by a stray dog which she was petting because it seemed to be sick. The dog was put out of the house and lost sight of. The wound was cauterized with nitrate of silver by a druggist and healed without suppuration. On 11th August the child complained of lassitude, but was restless, and on account of these symptoms her mother gave her a dose of castor oil. The next morning inability to swallow was observed, being attributed to "sore throat," and soon after her mother noticed that, on being touched with a wet towel or exposed to slight draughts of air, she was frightened and startled. Dr Krauss was called in on this day. The temperature was 100°, pulse 120. On giving her a glass of water she would look at it with dilated pupils for a moment, then suddenly grasp it and quickly attempt to swallow a mouthful. Every trial was followed by severe tonic convulsive contraction of the pharyngeal constrictors and the more external muscles of the neck, lasting about fifteen seconds. Repeated attempts were made, always with the same result. In reply to a question, she said that

she was not afraid of the water, but it hurt her throat very much when she swallowed it. She shrank from the slightest current of air. Large doses of bromide of potassium and chloral with morphin were given, but without effect, and the symptoms grew more marked. She passed a sleepless night. On the morning of the 13th her temperature was 101.5° , pulse rapid. Later in the day she developed a state of intense excitement. She would lie quiet and apparently conscious for a time, then without warning suddenly spring up to her mother, crouch down with short cries of fear, looking at the wall with an expression of great dread, and seemingly unconscious of her surroundings. After about two minutes she would wake up as from a dream. Repeated hypodermic injections of morphin had no effect, and she was sent to St. Christopher's Hospital. After a short remission the convulsive seizures became so frequent that almost constant inhalation of chloroform became necessary. When this was left off, she would utter short and loud cries of fear, which might well have been mistaken for the bark of a dog, and attempt to spring out of the bed. Death took place on the morning of the 14th at six o'clock. The *post-mortem* examination revealed congestion of brain and meninges and minute hæmorrhages, while the severity of the convulsions was proven by rupture of the pleura. The train of symptoms left no doubt in the mind of the attending physician that the case was one of hydrophobia, and the diagnosis has been abundantly confirmed by the inoculation of rabbits, as well as by microscopic examination of the bulb and plexiform ganglia of these animals. The inoculations were carried through four generations, the rabbits all dying with typical symptoms of rabies, with the exception of one of the first generation, which died thirty-six hours after inoculation from septicæmia. Besides the microscopic examination mentioned, full series of cultures were made with the object of detecting any accidental infection during inoculation or after. These cultures remained sterile in every instance, so that we are able to exclude with certainty any known bacterial disease.

Our work has led us also to the study of a number of control cases which are as yet incomplete. So far as we have gone, however, they induce us to believe that the lesions described by Van Gehuchten and Nelis may, for all practical purposes, be regarded as specific of rabies. We bear in mind the opinion of Van Gehuchten himself, and held also by some other observers, that in dogs at least the changes in the cerebrospinal and sympathetic ganglia are only specific of the natural or street rabies. The lesions produced by the experimental inoculation of the "fixed" virus are not as marked as those in natural cases, and at times may be completely absent. We have not experimented with a fixed virus, but in rabbits inoculated with street virus of the first and second generations the gangliar changes have been marked. We have been convinced that the discovery of Van Gehuchten and Nelis is of great importance, from a scientific as well as a humanitarian standpoint. It can be rapidly and easily carried out, and should be a matter of routine in the examination of suspected cases of rabies in every laboratory.

In the examination of the medulla several sections were taken at random for mounting, but serial sections, extensive investigations of different portions of the bulb were not made, inasmuch as we were

investigating rapid methods of diagnosis only, and not the essential nature nor the presence of these tubercles in the central nervous system. It is with this understanding that we recommend the examination of the ganglia rather than the bulb.

That these changes, or at least changes similar to them, might be expected in diseases where the intoxication is intense led us to inaugurate a series of experiments with tetanus and diphtheria toxins. In the meantime Crocq has reported his findings in the ganglia of a case of diphtheria, and since that time Spiller and Van Gehuchten have called attention to changes in three other cases, resembling those found in the ganglia in rabies. In Crocq's case the proliferation of capsular cells was present, but the absence of the perivascular changes, which are as diagnostic as the capsular changes, made the differentiation comparatively easy. In Spiller's first case, endothelioma of the Gasserian ganglion, the presence of the tumour mass causing the irritation leading to the capsular changes, and the slow course of the disease would lead to a correct conclusion. In the second case, one of meningomyelitis, the resemblance is very striking, but the capsular, interstitial, and pericellular changes are of such an intensity as is hardly met with even in the dog and horse, where the changes are much more intense than in man.

The attention which the subject has attracted, and the large number of investigations which are being made in consequence, will probably lead to the discovery of isolated cases here and there in which lesions closely resembling, or perhaps identical with, those under discussion will be found. So far only four of such cases have been reported, all in man. While these are sufficient to make us doubt the absolute specificity of the lesion, it can be said with certainty that neither in man nor in any of the lower animals is there any condition known in which these changes appear constantly except rabies. Their occurrence in this disease is so constant and so marked that we cannot but believe that they have great diagnostic value.

From our study of this subject the following conclusions seem justified:—

I. When present, the capsular and cellular changes in the intervertebral ganglia, taken in connection with the clinical manifestations, afford a rapid and trustworthy means of diagnosis of rabies.

II. That when these changes are not present it does not necessarily imply that rabies is not present. The lesions afford contributory evidence more or less valuable, depending on the duration of the clinical manifestations.

III. That in certain cases when the capsular changes are slight, such as in animals dying or killed in the early stages of the disease, the changes are more marked in the distal-peripheral end of the ganglion.

IV. That the rabic tubercle of Babes is present sufficiently often to furnish valuable assistance in cases where only the central nervous system is obtainable without any of the ganglia, but in cases where the ganglia can be had they offer a simpler and easier method of diagnosis than do the brain or cord themselves.

STREPTOTHRIX INFECTIONS IN THE LOWER ANIMALS.

By ALEXANDER G. R. FOULERTON, F.R.C.S., Lecturer on Bacteriology in the Middlesex Hospital Medical School, and C. PRICE JONES, M.B.

(From the Bacteriological Laboratory of the Middlesex Hospital).

Whilst our general knowledge of the natural history of the genus streptothrix has been considerably widened during the last few years by the careful study of different species, which have been isolated from cases of infection in man and lower animals, or have been found existing under saprophytic conditions, there is still very much to be done in the direction of exact differentiation of species, and correct observation of details of morphology and development.

Prior to 1875 nothing was known of this class of vegetable parasites. The older pathologists were familiar with the appearance of the tumours, occurring especially in connection with the tongue and lower jaw of cattle, which Leblanc in 1826 had described under the term osteosarcoma, but which are now known to be the result of an actinomycotic infection. Langenbeck as far back as 1845 had apparently noticed the presence in such lesions of certain bodies, which Lebert in 1857 also recognised in the pus of an abscess in the chest wall in man, and described as being of a crystalline nature. Rivolta in 1868 also identified similar bodies in osteosarcoma of the ox, which he regarded as being of a like nature; and Robin in 1871 again described as crystalline concretions what are now known as actinomycotic granules. Perroncito in 1875 recorded some observations on a fungus which he had found in cases of osteosarcoma in cattle, the organism being described as consisting of short branching threads with swollen or globular extremities. About the same time Rivolta renewed his investigation of these cases, and came to the conclusion that in such tumours there existed a parasite having the form of discoid granules composed of branching rods. Bollinger in 1877 published a further account of this parasite, and confirmed the observations of Perroncito as to its presence in the so-called osteosarcoma of cattle. Israel in 1878 recorded two cases of actinomycosis in man, and compared the disease with that occurring in the ox. Hartz in 1879 dealt with the parasite from the botanical point of view, and placed actinomyces bovis amongst the hyphomycetes, or mould fungi. Ponfick in 1882 maintained that the disease, by this time well recognised as actinomycosis in both man and cattle, was caused by one and the same parasite.

In 1884 Israel gave a description of the parasite when cultivated under artificial conditions—on coagulated ox serum—and his results were confirmed by Johne in 1885. In 1888 Nocard gave an admirable description of a vegetable parasite isolated from the lesions of a disease of cattle more especially common in Guadeloupe, and known as *farcin des boeufs*. The culture, morphology, and pathogenic action of the parasite were at once so fully described that but few lines can now, after a lapse of thirteen years, be added to Nocard's original paper. Nocard gives a description typical of an organism which would now be readily recognised as a streptothrix, but which

he himself then described as a fine, long bacillus occurring in the tissues of infected animals as a tangled mass of branching threads. During the last ten or twelve years a large number of other organisms of this genus have been isolated from various sources, and it is now very generally accepted that the actinomycosis of Bollinger and Israel is not a definite pathological entity, that it does not represent one specific infection, but that the word can be used merely as a generic term for an infection by one or other of the different species of streptothricheæ, or ray fungi, many of which show a strong mutual resemblance in their mode of pathogenic action.

In what follows we propose first to deal very briefly with the general characters of the streptothricheæ, and then to refer more especially to those species which have been found infecting the lower animals. Our general description of the characteristics of the genus is founded on a study of twenty-nine different species which we have been able to obtain from various sources, including eight apparently new species which we have ourselves isolated. Of these eight new species four have already been described,¹ and a description of the other four will shortly appear elsewhere.

NOMENCLATURE AND CLASSIFICATION.

The greatest possible amount of confusion has existed in the past with regard to the nomenclature and classification of the species of this genus. In the beginning the organism then known as *actinomyces bovis* was correctly placed by Hartz amongst the hyphomycetes, or mould fungi; but succeeding observers, not realising the true significance of the varying morphology of the fungus at different stages of development and sporulation, were led to class *actinomyces bovis* and similar organisms amongst the schizomycetes, or fission fungi. The varying morphology was explained by assuming that the organisms were capable of existing as either bacilli, spirilla, or cocci. Further error was introduced by a misunderstanding of the description given of the organism originally described as *cladotrix dichotoma* (*streptothrix nigrescens*). Owing to the use of the rather unfortunate expression pseudo-dichotomy, certain writers were led to believe that this organism described by Cohn showed a false branching, whereas it really shows a true, but not dichotomous, branching. *Cladotrix dichotoma* was accordingly placed together with other streptothricheæ in the genus *cladotrix*, all being regarded as fission fungi. At length, however, the confusion which has existed in respect of all this has been cleared up to the satisfaction of the majority of pathologists, and the streptothricheæ may be regarded as definitely assigned to their original place amongst the mould fungi.

The genus itself has been designated by different writers under the following terms: *actinomyces*, *cladotrix*, *oospora*, *nocardia*, and *streptothrix*. The designation *cladotrix* is misleading, and *oospora* appears to be incorrect, and has not been accepted by more than one or two of those who have studied these organisms; there is, therefore, no difficulty in discarding these two terms. The term *nocardia* one

¹ Foulerton on Streptothrix Infections, "The Lancet," 16th September 1899, and "British Journal of Dermatology," Vol. XI., 1899; Price Jones' Preliminary Note on Cultures of Streptothricheæ, derived from Various Sources, "Transactions of Pathological Society of London," Vol. LI., 1900.

would have liked to be able to retain in honour of a great pathologist, but as a matter of fact other terms have come into such general use that this one must be sacrificed. Consequently we are left to choose between *actinomyces* and *streptothrix* as the generic designation. Both terms are said to have been used in former times to designate fungi of a description different from those which we are now considering; but we do not think that this constitutes a practical objection to the use of either, as there is no chance of the former use of the terms being revived, and, indeed, there would probably be some difficulty in identifying the species which were formerly thus designated. Finally we have decided to adopt the term *streptothrix* in preference to the term *actinomyces*, especially because the latter has for so long been identified with one species as a specific name, that we think it would tend to perpetuate the confusion which lately prevailed in connection with these organisms if *actinomyces* were preserved as the generic name. Using then *streptothrix* as the generic designation of these fungi, the term *actinomycosis* still remains as a convenient general term, alternative to *streptothrichosis*, to describe an infection by any one of the *streptothricheæ*, or ray fungi.

With regard to the nomenclature of species again, a certain amount of difficulty has arisen; and in this case partly because of the wide variation in the appearance of cultures of the same species at different times and under but slightly different conditions, and partly because of the difficulty in the case of some species of getting any cultures at all. Owing to these circumstances, there is no doubt but that the same species have been described under different names by writers who when dealing with them were not aware that they had already been described.

MORPHOLOGY AND STAINING CHARACTERISTICS.

The morphology of this group of parasites was worked out fairly accurately at an early period, and much of the confusion which has since arisen has resulted from the efforts of some of the later writers on the subject.

If one takes a sporulating culture of a quickly growing *streptothrix*, and incubates it in a hanging drop culture on a warm stage at 37° C., one sees at the end of twenty-four hours that an obvious sprouting from the spores has occurred, either one or two threads springing from each spore. These threads then produce lateral branches by a process of budding. The later morphology of the mycelium thus produced can be studied in later stages in cultures on various media. In general the mycelium appears as a mass of branching threads, the extremities of which, and more especially the extremities of the side branches of the parent trunk, may be either pointed or terminate in small globular or elongated swellings. After a time shorter or longer lengths of the mycelium, here and there, become altered in character. This alteration in character is manifested by an alteration in the staining properties of the part concerned. The altered portions are stained much less deeply by aqueous solutions of basic anilin dyes than formerly, and they have lost the power of retaining the gentian violet stain when treated by Gram's method. When stained, moreover, they appear of less thick-

ness than the intervening normally staining portions of the mycelium. This alteration of the staining properties of portions of the mycelium appears to us to mark the commencement of a process of fragmentation by which the mycelial thread is broken up into longer or shorter segments. This process of fragmentation by a degeneration and disappearance of definite lengths of the mycelium must be clearly distinguished from the clean fission, without obvious loss of substance, by which reproduction occurs amongst the schizomycetes. As a general rule, what may be termed "chain sporulation" is in evidence as a commencing process nearly coincidently with the commencement of the process of fragmentation. The first manifestation of chain sporulation, as we have observed it, is indicated by the appearance as of a beading at the terminal portions of the mycelial threads. On staining by Gram's method it is seen that these terminal portions present the appearance of being composed of alternate small segments which either have or have not retained the stain. The portions of the beaded terminal segments which retain the stain subsequently, and after the disappearance of the intervening degenerated portions, become free spores. These free spores under favourable conditions give rise to a new generation of mycelium, and the cycle of development and sporulation is under favourable circumstances again traversed. Returning to the more central portions of the mycelium which is undergoing a fragmentation by the process already described, we find that in the result a number of shorter or longer segments of the original mycelium appear as free elements, intervening portions of the original mycelium having degenerated and disappeared, just as do the portions of mycelium which intervene between the spores. These free segments retain the normal staining properties of the original mycelium, and in some species acquire further what are known as acid-fast properties, that is to say, when stained by Ziehl-Neelsen method they do not yield up the carbol-fuchsin when washed with 33 per cent. solution of nitric acid in water. Some of the species which we have studied still retained the stain when subsequently washed for ten minutes in absolute alcohol. The ultimate fate of these persistent segments is not quite certain; we think that when transplanted to a fresh medium they are certainly capable of producing fresh mycelium by sprouting, just as do the spores. One thing, however, is quite certain, and that is that these short segments never reproduce similar short segments without a previous development of a mycelium. We also think it probable that if under artificial conditions these persistent segments do not gain access to a fresh medium they undergo a process of chain sporulation similar to that which takes place in the original mycelium. Space does not permit us now to enter more fully into this part of the subject; we can only point out that M'Fadyean¹ in 1889 gave a very full account of the morphology of the parasite as it occurs in the commoner actinomycosis of the ox, and that Domec² clearly enunciated the reasons for regarding the spherical bodies met with in cultures of streptothricæ, and also in infected tissues, as being true spores, and not of the nature of

¹ J. M'Fadyean: *The Morphology of the Actinomyces*. "Brit. Med. Journal," 1889, Vol. I., pp. 13-39.

² Th. Domec: *Contribution à l'Etude de la Morphologie de l'Actinomyces*. "Archiv. de Méd. expér. et d'Anat. pathologique," Tome IV., p. 104, 1892.

cocci. Domec carefully observed the changes of development which occur in hanging drop culture, and reproduced in his paper a drawing almost identical with the one which we give here as an actual representation of what occurs under the conditions.

In concluding this part of our subject we can now merely point out the fact that the more one studies these organisms the more one is struck by the affinities which the bacillus tuberculosis of Koch presents to the members of this genus, and we think that the arguments which



Hanging drop sub-culture in broth of streptothrix from pure spore culture, twenty-four hours on warm stage. Drawn under Zeiss one-twelfth oil immersion lens, eye-piece No. 4.

several have urged in favour of classifying this latter organism with the streptothricheæ will in all probability be justified by the results of further research work.

CULTURAL CHARACTERISTICS.

Space, again, will not allow us to deal in more than the briefest possible manner with the cultural characteristics of the genus. Details as to the more important species affecting lower animals will be found in the description given at the end of the paper.

The majority of the species appear to be facultative ærobes, and with regard to streptothrix capræ, and streptothrix nocardii in particular, our experiments do not justify the accepted statements as to these being more or less strict ærobes. As to pigment formation on various media, certain species show the greatest irregularity in this respect, and we are not at all inclined to accept any classification of the species founded on so uncertain a property. With regard to growth on gelatin at 22° C., this occurs on all cultures which we have had the opportunity of fully examining, but in most of the species growth is more luxuriant at 37° C. than at the lower temperature. We have examined only one species so absolutely saprophytic that growth does not occur at animal blood temperature. As a rule species grow in rather scanty fashion on a purely proteid medium, such as blood serum. Growth in milk may or may not be accompanied by obvious changes in the medium; the commonest change is manifested by a gradual clearing up of the normal opacity of the medium, without a preliminary coagulation, such as usually happens with bacteria. The nature of this particular change is not obvious, but it appears to us to be of the nature of a saponification rather than a peptonisation. As to enzyme secretion, we have obtained certain data for the species which we have investigated. Some manifest an active diastatic action, converting starch readily into sugar; others liquefy gelatin without peptonising blood serum; and others peptonise both media. Growth on potato shows in a high degree the variability in pigmentation production which characterises many of these species; and on this medium the formation of ærial hyphæ with consequent sporulation is as a rule especially rapid. Growth in broth has the common characteristic that the greater part of the growth takes place at the bottom of the tube in the form of small discoid colonies, which may remain discrete or may form a more or less closely coherent mass. Growth in the form of a surface veil, with the formation of ærial hyphæ, occasionally takes place, and occasionally small discoid colonies may be seen adhering to the side of the culture tube, but the rule that the bulk of the broth remains quite clear and free from growth appears to be invariable.

PATHOGENIC ACTION, AND ANIMALS AFFECTED.

Using the word in its earlier sense, as it was used by Bollinger and Israel, Nocard writes¹ of actinomycosis as occurring in the ox, the horse, the pig, and more rarely, in the sheep. He also quotes Schreiber as having observed actinomycosis of the liver in the stag, and Monfallet as having met with actinomycosis of the jaw in the lama and guanaco of Chili.

The fact must, however, be again insisted on that the term "actinomycosis" now merely implies an infection by one or other of the streptothricheæ, and whilst for this purpose it is a convenient enough expression for general use, it can no longer be used for exact pathological purposes to denote an infection by the one specific organism, streptothrix bovis communis (syn. actinomyces bovis); and it is not by any means certain what proportion of the cases upon which

¹ "Les Maladies microbiennes des Animaux." Paris, 1898.

data have hitherto been founded really represent infections by this one particular parasite.

In the case of the ox, there is the actinomycosis of Bollinger caused by *streptothrix bovis communis*, and there is the actinomycosis known as farcin des bœufs and caused by *streptothrix nocardii*. The clinical manifestations of these two diseases are quite distinct, and the respective parasites are readily differentiated. The parasite of the former disease infects more commonly by the upper part of the alimentary canal—by the mouth and pharynx—less frequently by the air passages, and again much less frequently by the lower part of the alimentary canal beyond the stomach. The resultant lesions usually take the form of single "tumours," which may attain to a considerable size, and which, in a large proportion of the cases are situated in connection with the maxilla, in the substance of the tongue, or towards the upper part of the neck. Next, in frequency of occurrence, probably come nodules of larger or smaller size in the air-passages; whilst the appearance of nodules in the abdominal glands or viscera is rare, and in the bones, other than those of the face, more uncommon still. *Streptothrix nocardii* usually infects by some accidental cutaneous lesion, and in most cases a chronic, very slowly extending infection of the lymphatic vessels of the subcutaneous connective tissue and the deeper lymphatic glands is the result. The clinical manifestation of this infection is the development of a series of, at first, hard swellings along the course of the superficial lymphatics and in the situation of the deeper glands of the limbs; these swellings may then so increase as to form firm, subcutaneous tumours of a considerable size, or, on the other hand, may break down and suppurate. In some cases the lungs, liver, and other viscera may in time become infected.

Gasparini recognises, besides *streptothrix nocardii*, three different species as infecting the ox—*actinomyces sulphureus* (syn. *actinomyces bovis*), *actinomyces albus* (syn. *streptothrix* 1, 2, 3 of Almquist, and *streptothrix alba* of Rossi Doria), and *actinomyces luteo-roseus*.

In the pig also, at least two actinomycoses have been described. In one, the parasite is apparently the same as that causing the commoner infection of the ox; and in this case the lesions have very much the same character and distribution as in the latter animal. In the other infection, the lesions are situated exclusively in the muscles, and a causative parasite, *streptothrix musculorum suis* (syn. *actinomyces musculorum suis*, Dunker, Hertwig) has been described by Hertwig,¹ who, however, apparently founded his study of the parasite on microscopic examination of the infected muscles, no cultures—as far as we can ascertain—having been obtained. Our knowledge of this parasite is, therefore, deficient; and, indeed, the fact of these lesions in the muscles of swine being caused by a ray fungus at all, has been questioned, and is, as a matter of fact, extremely doubtful, Olt stating that they are caused by a streptococcus, and Zimmermann believing the parasite to be an animal one, a species of sarcosporidia.

As to streptothricial infections in the horse we have not been able to obtain much definite information. Actinomycoses are of fairly common occurrence in this species, but apparently the causative

¹ Ueber den *Actinomyces musculorum der Schweine*. "Archiv für wiss. und pract. Thierheilkunde," Bd. XII, 1886.

parasites have not been isolated and studied to any purpose. The lesions occur in much the same situations, and the disease runs very much the same course as in the commoner form of actinomycosis occurring in the ox.

Dr Dean has studied¹ a streptothrix, which came from an abscess situated near the angle of the jaw in a horse. The organism was rather difficult of growth, but cultures were obtained on agar, in peptone beef broth, and in Spronk's broth. No growth could be obtained on gelatin, potato, egg albumin, coagulated blood serum, or in milk. Growth under anærobic conditions was very scanty. On agar, colonies appeared after from three to five days' incubation at 37° C., the growth assuming the appearance of small masses of white coral. No pigment was formed at any time during the growth of the cultures. The cultures were pathogenic for several of the species inoculated; small nodules containing the parasite resulted after inoculation in nine out of seventeen rabbits, in two out of eight guinea-pigs, and in one of two pigeons. Four mice which were inoculated showed no signs of infection.

Dr Dean considers that his organism was a streptothrix, and so far we entirely agree with his interpretation of his experiments. But he appears to us to be under an erroneous impression as to the so-called "pleomorphic" nature of the streptothricheæ. Thus, Dr Dean writes of "the bacillary form which persists on agar, etc.," and considers that the "streptothricheæ are more nearly related to the bacilli, such as the diphtheria and tubercle bacilli, than to the moulds." We venture to think that a careful study of the characters of the streptothricheæ under artificial cultivation would leave no doubt as to the very clear distinction which can be drawn between these and the typical fission fungi. The position of the reputed bacilli—*bacillus diphtheriæ*, *bacillus mallei*, and *bacillus tuberculosis*—has, on the other hand, been a source of doubt to many pathologists for some time past. But the results of more recent research have led in the direction of placing these reputed bacilli, or, at any rate, some of them, in the genus streptothrix, rather than in that of reducing the streptothricheæ to classification amongst the fission fungi. The question now is, not whether the streptothricheæ should be placed with the fission fungi, but whether certain reputed fission fungi—and of these more especially *bacillus tuberculosis*—should be placed with the streptothricheæ amongst the mould fungi.

Schmorl has described a streptothrix fungus which was isolated from the lesions of an epidemic disease which occurred amongst some rabbits at the Pathological Institute at Leipzig. Twenty-five of the animals, which occupied one particular pen, died, and from their organs a streptothrix was isolated in pure culture in almost every case. When inoculated subcutaneously into other rabbits, the parasite produced lesions similar to those found in the natural infection, that is to say, a fibrinous inflammation of the serous membranes of the peritoneum of the pleuræ, and of the pericardium.

In dogs an actinomycosis has been described by Rabe, which appeared in two cases as a "suppurative phlegmon," and in another as a peritonitis. The description of the fungus present—streptothrix *canis* (syn. *cladothrix canis*, Rabe)—is rather vague, but Gasparini

¹ "Trans. of the Path. Soc. of London," Vol. LI., 1900.

and others identify it with *discomyces pleuriticus canis familiaris* of Rivolta¹ and Vachetta. It is not certain how far this statement of the identity of the two parasites is correct, as another organism apparently of the same class—*discomyces equi*—described by Rivolta as the cause of botryomycosis or discomycosis of the cord in castrated horses is, according to Johne, Rabe, Bollinger, and M'Fadyean,² a *staphylococcus*.

Terni has isolated from lizards an organism, *streptothrix lacertæ*, which is found in the liver of the reptile, and reproduces the infection when inoculated into healthy lizards.

Speaking generally, there is a strong similarity between the pathogenic action of these various *streptothricheæ*. The common result of infection is the formation of granulomata wherever the parasites come to rest in the tissues. In these granulomata the fungi pass through a regular course of development, the mycelium subsequently undergoing fragmentation, and sporulation in some cases occurring. The diffusion of infection in the body must be effected in the majority of cases by the spore forms. The parasite does not, as a rule, seem to have an actively toxic local action on the tissues, although several of them secrete peptonising enzymes, and one or two species appear to be primarily pyogenic. The granulomata formed are of the usual histological type, and may or may not show the presence of giant cells. They increase in some cases to a considerable size, without undergoing much structural change, or, on the other hand, they may undergo caseous degeneration, with or without subsequent breaking down of the degenerated granulomata tissue, and the formation of purulent fluid. Fibrinous inflammation of the serous membranes appears to be another occasional result of infection. The *streptothricheæ* do not, as a rule, appear to secrete substances actively toxic to the infected organism generally.

DESCRIPTION OF SPECIES.

The following description of species is taken from our own observations in the cases of *streptothrix bovis communis*, *streptothrix nocardii*, and *streptothrix capræ*. The description of the first species is founded on the examination of several cultures, which we have been able to obtain from different laboratories. Two cultures of *streptothrix nocardii* have been examined, one an old laboratory culture, the other, one which Nocard himself was so kind as to give us. Our culture of *streptothrix capræ* was obtained from Kral of Prague. Whenever there has appeared to be any discrepancy between our own observation and that of the original writer, the fact has been duly noted. In the case of the two last species on the list, we have not been able to obtain cultures for ourselves, but give as full a description as we can find available in the writings of others.

(1) *Streptothrix bovis communis*.

Synonyms.—*Streptothrix bovis*, *streptothrix actinomycotica*, *actinomyces bovis*, *actinomyces bovis sulphureus*.

¹ Del micelio e della varieta e specie di *Discomiceti* patogeni. "Gior ali d. Anat. Fisiol. e Patol.," XVI., 1884.

² *Metastatic Lesions in Discomycosis*. "Journal of Comparative Pathology," Vol. XIII., 1900.

Described by Perroncito, Rivolta, Bollinger, Hartz, and Israel.

Occurs in the lesions of the common actinomycosis of the ox, and to an at present unascertained extent in other herbivora.

Morphology.—Mycelium appears as a mass of branching threads, which even in old cultures (four months) on artificial media show less tendency to fragmentation than many of the other streptothricheæ. The side branches are numerous, and usually short, often terminating in swollen extremities; these side branches frequently appear of greater thickness than the parent stem. In old cultures (four months in broth, and two months on potato) threads showing regular, small beading are infrequent, and free spores are few in number.

Staining Characteristics.—In a general way this species stains according to the rule given above, but in some quite young cultures the mycelium will not stain by Gram's method. In none of the cultures which we have examined do any "acid-fast" segments appear, even in very old cultures.

Cultural Characteristics.—Growth occurs under ordinary aerobic conditions, and in vacuo.

On Gelatin at 22° C.—Streak culture: growth appears after about ninety-six hours' incubation, and presents a yellowish white appearance. A slow liquefaction of the medium occurs, and is first obvious about the seventh day. In stab culture: growth occurs along the track of the stab in the form of a series of opaque, whitish, super-imposed discs, which diminish in circumference from near to the surface downwards. Liquefaction takes place slowly, the surface of the liquefied gelatin at first supporting a tenacious film of the growth, which afterwards sinks. No pigmentation of the gelatin takes place to any noticeable extent.

On Maltose Nutrient Agar at 37° C.—Growth occurs as small, fawn-coloured discrete colonies, after forty-eight to seventy-two hours' incubation. The surface of the growth has, at first, a smooth, waxy appearance, but afterwards, becomes dull, and changes to a canary yellow, dark brown, or almost black, colour.

On Inspissated Horse Serum at 37° C.—Growth is slow, and not extensive at any time. No liquefaction of the medium occurs.

On Potato at 37° C.—Raised irregular growth, the appearance of which varies in different cultures from the same stock, appearing at one time of a brownish, at another of a light canary yellow, at another of a greyish black, or quite black, colour. The potato becomes in time more or less eroded, and sometimes deeply pigmented.

In Peptone Beef Broth at 37° C.—Growth of whitish, opaque colonies, either discrete, or more or less closely coherent, and often showing a slightly pigmented centre. Surface growth apparently but rarely occurs. The broth is not perceptibly pigmented.

In Alkaline Litmus Milk at 37° C.—During the first ten days or so of incubation no obvious change occurs in the milk; later the medium is slightly pinkened, and the colour discharged. Meanwhile, about the fourteenth to the eighteenth day, the milk begins to clear from above downwards, ultimately appearing as a brownish, transparent fluid.

No diastatic action is manifested after fourteen days' incubation in starch broth at 37° C.

Resistance to Heat.—Cultures containing spores resist exposure to

60° C. for thirty minutes, but are destroyed by an exposure to 75° C. of the same duration.

Pathogenicity.—The pathogenicity of the streptothrix for the ordinary laboratory animals does not seem to have been thoroughly worked out; it is, however, pathogenic for the guinea-pig, producing a nodular infection, in the lesions of which the parasite may be demonstrated.

(2) *Streptothrix Nocardii.*

Synonyms.—*Streptothrix farcinica*; *actinomyces farcinicus*.

Described by Ed. Nocard: "Note sur la Maladie des Boeufs de la Guadeloupe connue sous le nom de Farcin," *Ann. de l'Institut Pasteur*, tome ii., p. 293, 1888.

Occurs in lesions of farcin des boeufs.

Morphology.—Mycelium appears as a mass of branching threads of unequal thickness, with rather infrequent branching. In stained specimens from young cultures (twenty-four hours old in peptone beef broth at 37° C.), the threads sometimes show a fine granular appearance, whereby their contour loses the sharply defined outline seen in most other species. In older culture (forty-eight hours) many of the filaments already show regular, small beading, and a few small spores may be seen. In still older cultures (eight days, culture as before), the mycelium has undergone complete fragmentation, and is represented by a few beaded threads; there are numerous free spores.

Staining Characteristics.—In quite young cultures the mycelium stains throughout its length with aqueous solutions of the ordinary anilin dyes. In older cultures, when fragmentation of the mycelium is in progress, short lengths of the threads here and there stain only faintly, and appear less thick than the remaining deeper stained portions. Stained by Gram's method, in young cultures the mycelium retains the gentian violet stain throughout. In older cultures those portions of the length of mycelium which stain faintly with the aqueous stains are found to have lost the power of retaining the gentian violet. The spores stain by Gram's method. Stained by the modification of the Ziehl-Neelsen method which we have employed, in young cultures only a very few isolated portions of the mycelium are acid-fast. In cultures of from nine days to three months age a relatively large proportion of the growth is acid-fast, the colour not being discharged by a subsequent washing in absolute alcohol for ten minutes.

Cultural Characteristics.—Growth occurs under ærobie conditions, and also in vacuo (strict ærobe, will not grow in atmosphere of carbon-dioxide or in vacuo.—Nocard).

On Gelatin at 22° C.—Streak culture; growth appears on fourth day as a fine white opacity along streak, with an uneven surface; no liquefaction of the gelatin occurs. In stab culture there is a slightly raised knob at upper end, and a fine filmy growth along whole length of stab. Growth at this temperature remains scanty.

On Maltose Nutrient Agar at 37° C.—Pale, straw-coloured growth, with dull, flattened granular surface, appears after forty-eight hours' incubation.

On Inspissated Horse Serum at 37° C.—Growth very slow; no liquefaction of medium.

On Potato at 37° C.—Buff yellow, granular-looking growth ; no obvious erosion of the potato.

In Peptone Beef Broth at 37° C.—Greyish flocculent mass of growth collects at the bottom of the broth (growth does not alter the reaction of neutral or alkaline broth, even when the latter contains glucose. —Nocard).

In Alkaline Litmus Milk at 37° C.—Growth occurs ; reaction and appearance of milk not obviously altered after twenty days' incubation.

No pigmentation of the medium appears in any of the above cultures.

No diastatic action is apparent after fourteen days' incubation in starch broth at 37° C.

Resistance to Heat.—Cultures containing spore forms resist exposure to 45° C. thirty minutes, but are killed by an exposure to 60° C. of the same duration (cultures resist exposure to 65° C. for fifteen minutes, but are killed by exposure at 70° C. for ten minutes. —Nocard).

Duration of Vitality in Cultures.—Cultures remained virulent after four months' incubation at 40° C. (Nocard).

Pathogenicity.—Rabbits, dogs, cats, horses, and asses are not affected by either intra-peritoneal or intra-venous injection. In guinea-pigs intra-peritoneal or intra-venous injection causes constantly, within nine to twenty days, a miliary tuberculosis similar to that produced by bacilli tuberculosis. The abdominal viscera are widely invaded, the thoracic viscera never. In the rabbit, dog, horse, and ass inoculation into the subcutaneous tissue causes a local abscess which discharges itself and heals. In the guinea-pig, the sheep, and the ox an ulcer remains at the site of inoculation, and a lymphatic infection follows. In guinea-pigs the course of the disease was comparatively slow ; one animal died after fifty-four days, and fifteen killed between one and five months after inoculation showed no lesions beyond those in the lymphatic vessels and glands in the neighbourhood of the site of inoculation.

In the lymphatic abscess numerous organisms which had preserved their normal virulence were found. In sheep and in oxen a series of abscesses developed about the site of inoculation ; but the disease was, as in the natural conditions when the animal may live for several years, an extremely chronic one. The organism remained localised in the lymphatics, and showed no tendency to cause a general infection. (This account of the pathogenicity is abstracted from Nocard's paper).

(3) *Streptothrix Capræ.*

Described by Silberschmidt : " Sur un nouveau Streptothrix Pathogène (*Streptothrix Capræ*)," *Ann. de l'Institut Pasteur*, tome xiii., p. 841, 1899.

Occurred in the nodular lesions of the lung of a she-goat.

Morphology.—The regular mycelium of young cultures soon breaks up into short beaded segments, and other short segments which are of homogenous structure throughout, and which sometimes show a small globular swelling at the extremity. Free spores occur early. In old cultures (six weeks incubation on maltose agar at 37° C.) the

mycelium is replaced entirely by free spores, short chains of spores, and short straight segments lying either singly or arranged in short chains, with small spaces between successive elements.

Staining Characteristics.—In young cultures all portions of the mycelium stain by Gram's method; in older cultures the spores and separate segments take the Gram staining fairly evenly. In old cultures many segments when stained by the modified Ziehl-Neelsen method are acid-fast, and retained the carbol-fuchsin after a subsequent washing in alcohol for ten minutes.

Cultural Characteristics.—Growth occurs under ordinary aerobic conditions, and also in vacuo (essentially aerobic, grows not, or scarcely at all, in the absence of oxygen.—Silberschmidt).

On Gelatin at 22° C.—Streak culture: growth appears on third day, and develops as a very slightly raised layer, the surface of which is covered with a chalky efflorescence, and has a faint pinkish tint. Viewed from the under surface of the gelatin the growth has a wrinkled appearance, and a more pronounced pink tinge. No liquefaction or pigmentation of the medium occurs (colonies on gelatin have a brownish centre with clearer periphery.—Silberschmidt). In stab culture a small chalky knob develops at upper end, and a fine filmy growth, which towards the upper end has a pinkish tint, occurs along track of stab.

On Maltose Nutrient Agar at 37° C.—Free, spreading, drab-coloured growth occurs; surface in parts dull and with powdery appearance; slight darkening of the medium is seen in old cultures. Growth is obvious after twenty-four hours' incubation (colonies have an irregular surface, and after eight to fifteen days' incubation are of a light brown colour, and look as if powdered over with a white, starchy layer.—Silberschmidt).

On Inspissated Horse Serum at 37° C.—Free growth occurs; there is no liquefaction of medium (the culture is like that of the bacillus of human tuberculosis.—Silberschmidt).

On Potato at 37° C.—A slightly raised, finely granular growth, of brown to grey colour, partly covered by chalky-looking efflorescence; the potato is tinged brown, and no obvious erosion of it occurs (on potato growth is of a "brun rose" colour, which is less pronounced when the tube is closed with rubber cap.—Silberschmidt).

In Peptone Beef Broth at 37° C.—Growth appears as rather small isolated colonies, and flocculent masses of coherent colonies; growth occurs at surface as well as at bottom of broth.

In Alkaline Litmus Milk at 37° C.—After twenty days' incubation the milk shows no change beyond a very slight pinking, growth on surface of the milk (after several days a film of a "blanc rose" colour appears on surface of milk; coagulation does not occur.—Silberschmidt).

No diastatic action is apparent after fourteen days' incubation in starch broth at 37° C.

Resistance to Heat.—Cultures containing spore forms resist exposure to 60° C. for thirty minutes, but are killed by an exposure at 75° C. of the same duration. (Cultures are killed by exposure to 70° C. for sixty minutes, and by an exposure to 80° C. for twenty minutes; lower limit not ascertained.—Silberschmidt.)

Vitality of Cultures.—Cultures exposed in the laboratory to diffused light were successfully subcultured after seven months.

Pathogenicity.—(Silberschmidt.) In rabbits inoculation into the subcutaneous tissues produced a local abscess; intravenous inoculation produced nodules in various organs; the pulmonary nodules present giant cells, and caseate rapidly. Four mice were inoculated in the subcutaneous tissue: two of these suffered nothing from the inoculation, and in the other two local abscesses formed, from one of which the streptothrix was recovered. Two mice received intraperitoneal injections, and remained well. In the guinea-pig subcutaneous inoculation in one case was followed by death on the eighteenth day; there were numerous suppurating nodules in the parietal peritoneum, the mesentery, the diaphragm, the liver, kidney, and spleen. From these nodules cultures were obtained. Two other guinea-pigs recovered with a local abscess after a similar inoculation, and one died with a large abscess at the site of inoculation, but no other lesions. Three guinea-pigs received intraperitoneal injections: one recovered, the other two died on the sixth and thirty-first days respectively. In these there were found very numerous miliary nodules on the diaphragm, on the surface of the liver, mesentery, serous coat of stomach, and on the testicles.

(4) *Streptothrix Cuniculi*.

Synonym.—*Actinomyces cuniculi*.

Described by G. Schmorl: "Ueber eines pathogenes Fadenbacterium." *Zeitschrift für Thiermedizin*, 1891. Ref. *Centralblatt für Bakteriologie*, erste abt., band xi., p. 666.

Occurred in lesions of an epidemic disease amongst laboratory rabbits.

Morphology.—Described as a "thread" bacterium, belonging to class of cladothriceæ.

Cultural Characteristics.—Is an obligatory anærobe, and cultures were obtained only on blood serum.

Pathogenicity.—In the natural infection the disease started about the mouth and spread rapidly, causing a necrosis of the subcutaneous tissue, with a fibrinous inflammation of the pleuræ, pericardium, and peritoneum, together with inflammatory changes in the lungs. The organism was found in the organs, mostly in pure culture, in every case. Inoculated into healthy rabbits it produced a similar disease, and was recovered from the resulting lesions.

The white mouse is susceptible in much the same way as the rabbit; guinea-pigs, dogs, cats, pigeons, and fowls were immune against infection.

Note.—Lachner-Sandoval and others consider that this parasite is identical with the organisms described by Loeffler as the bacillus of calf-diphtheria, by Flügge as bacillus diphtheriæ vitulorum, and by Bang as the necrosis bacillus.

(5) *Streptothrix Lacertæ*.

Synonym.—*Actinomyces lacertæ*.

Described by C. Terni: "Actinomicosi della lacertola, *Actinomyces lacertæ*." *L'Ufficiale Sanitario*, 1896. Ref. *Centralblatt für Bakteriologie*, erste abt., band xix., p. 953.

Occurs in greyish nodules in liver of *lacerta agilis* and *lacerta viridis*.





Fig. 1.

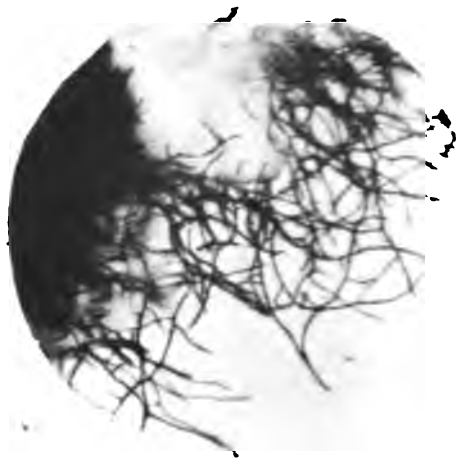


Fig. 4.



Fig. 2.



Fig. 5.



Fig. 3

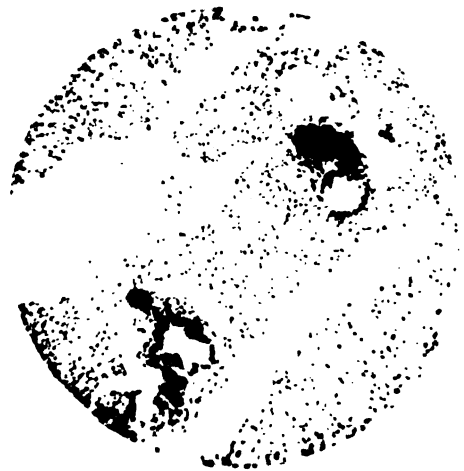


Fig. 6.

Morphology.—Described as an anctinomyces.

Cultural Characteristics.—Gives yellowish growth on glycerin-agar.

Pathogenicity.—On inoculation into lizards (*lacerta agilis*, *lacerta viridis*, and *lacerta muralis*), and into *coluber viridi flavus*, the parasite produced nodules in the liver.

Galli-Valerio (*Il moderno Zooiatro*, 1897) has found streptothricheæ in the liver of lizards; and it is suggested that by these lizards streptothricheæ may be distributed over pastures (Nocard).

DESCRIPTION OF FIGURES, PLATE I.

FIG. 1. *Streptothrix Nocardii*, edge of colony from broth culture twenty-four hours old ($\times 750$).

FIG. 2. *Streptothrix Nocardii*, edge of colony from broth culture forty-eight hours old ($\times 750$).

FIG. 3. *Streptothrix Nocardii* from broth culture six weeks old ($\times 750$).

FIG. 4. *Streptothrix Capræ*, edge of colony from broth culture twenty-four hours old ($\times 750$).

FIG. 5. *Streptothrix Capræ* from broth culture six weeks old ($\times 750$).

FIG. 6. *Streptothrix Bovis Communis* in nodule of tongue ($\times 750$).

(Photographs by Mr J. Barnard.)

EDITORIAL ARTICLES.

THE DEATH OF QUEEN VICTORIA.

FOR the citizens of the British Empire the dawn of a new century has been clouded by the sorrow of a great bereavement. On the 22nd of January last the hand of death touched our great and good queen, and removed from the scene of her earthly activity the best-beloved monarch the world has ever seen. It is no exaggeration to say that the national grief created by the event had an intensity bordering on stupefaction ; and, although with the lapse of time the oppression of our sorrow is passing away, we are daily realising more fully the magnitude of the nation's loss and the grandeur and beauty of the life that has been brought to a close.

In this brief page we do not set before ourselves the ambitious task of tracing, even in barest outline, the manifold graces and virtues of our departed sovereign, nor do we care to attempt to analyse the qualities and circumstances which drew to her the admiration and affection of the countless millions of her subjects, irrespective of creed, colour, calling, or station in life. But, venturing to speak on behalf of the profession to which we belong, we desire to place on record here our sense of participation in the great national loss, to add our tribute of admiration for a life which will endure as an example to the highest and the lowest, and to express our feelings of sympathy, loyalty, and devotion to our new sovereign, King Edward VII.

THE POST-MORTEM DIAGNOSIS OF ANTHRAX.

A QUESTION of considerable interest to veterinary surgeons, and especially to those who are engaged in what may be called country practice, is discussed in the article from the pen of Mr Reeks, which appears at an earlier part of this number of the *Journal*, viz., the procedure which the practitioner ought to adopt when he is confronted with a dead animal of the ox species, and there are reasonable grounds for suspecting that the cause of death may have been anthrax. As Mr Reeks observes, the first question which presents itself in such circumstances is: Shall a *post-mortem* examination be made? By *post-mortem* examination is here meant an operation involving the opening of the great body cavities and the complete or partial evisceration of the carcass. The author of the article answers this question with an emphatic "No," and he hazards the opinion that at least with regard to that point everyone will agree with him. We wish it were possible to believe that there is no diversity of practice in this matter among the members of the profession, but, unfortunately, our experience proves that there is, and that the skinning and disembowelling of cattle and other animals suspected of having died from anthrax, before any microscopic examination has been made, is by no means a rare occurrence even at the present day. As Mr Reeks says, the procedure is one that verges on criminality, and the well-informed must wonder why anyone should ever be guilty of it. Probably in most cases of the kind the guilty person, when arraigned, could truthfully put forward a plea of ignorance, for as recently as ten years ago the practice of making a more or less complete *post-mortem* examination in suspected cases of anthrax was almost general, and we fear it must be confessed that a not inconsiderable number of the older members of the profession take little trouble to keep themselves informed with regard to the modifications of practice which are almost daily being imposed by new discoveries in the realms of pathology and etiology. Incredible as it may appear, there are still to be found veterinary surgeons who in cases of suspected glanders, find their chief aid to diagnosis in dropping some of the nasal discharge into a bucket of water, and observing whether it sinks or swims. In like manner, the sister profession still includes venerable family physicians who consider heredity the only cause of human consumption, and who are scarcely ever apprehensive of an attack of tetanus unless the thumb or the great toe has been wounded.

It is probable, however, that most cases in which animals suspected of anthrax are eviscerated admit of another explanation. It is not unlikely that in a good many instances an ordinary *post-mortem*

examination is made deliberately, and in full consciousness of the risks which are inseparable from it, because the operator knows that he cannot rely upon his own interpretation of the microscopic appearance of a specimen of the suspected blood, and, with a view to the avoidance of delay, desires to see immediately the organs whose appearance he can trust to guide him to a correct conclusion.

Putting aside for the moment the objections to this method in the shape of risk to the operator, and the inevitable contamination of the ground, it raises the question of the reliability of macroscopic appearances in the diagnosis of anthrax. As a point in favour of the practice, it must be admitted that in the species which furnishes the largest number of cases of anthrax in this country the risk of error is not very great when the diagnosis is based on the condition of the internal organs. In the ox, the swelling and softening of the spleen, the intense congestion and enlargement of the mesenteric or other abdominal lymphatic glands, and the hæmorrhagic gastro-enteritis, make up a combination that is very seldom encountered in any other disease than anthrax. In the horse also, the lesions found within the abdomen in anthrax are tolerably characteristic, though the enlargement and softening of the spleen are not as a rule so pronounced in this species as in cattle. On the other hand, a diffuse, non-traumatic swelling in the region of the throat is, when present, an important guide to diagnosis in the equine species. As is well known, this last condition is present in the majority of cases of anthrax in the pig, and when, at the same time, there is a history of the animal having been recently fed with the raw flesh of some other farm animal unexpectedly found dead, the case may with scarcely any risk of error be diagnosed as one of anthrax. Undoubtedly, the animal in which macroscopic appearances are least reliable in the diagnosis of anthrax is the sheep. In that species the spleen is often but little altered in size or appearance, and the other lesions within the abdomen are often seriously masked by the putrefactive changes which have taken place before the *post-mortem* examination is made. For these reasons, the diagnosis of anthrax in sheep is little better than guess-work, when it is not supported by bacteriological evidence.

Mr Reeks, although an advocate for microscopic examination in all suspected cases, admits that even without it a practitioner might arrive at a correct conclusion in ninety-nine cases out of a hundred. That, we think, is much too favourable an estimate of the trustworthiness of naked-eye appearances and ; in our view, the practice of eviscerating animals suspected of anthrax deserves to be condemned because the outcome of it is nearly always uncertainty, and frequently error, and because an alternative method that is perfectly practicable conducts to almost absolute certainty in diagnosis.

We are, therefore, in agreement with Mr Reeks when he contends

that in every case of suspected anthrax resort should be had to microscopic examination of the blood. We are not so sure, however, that he is giving sound advice when he counsels the veterinary surgeon who is not an expert microscopist to base his diagnosis mainly on the microscopic examination of a stained and mounted specimen of the blood. We are quite prepared to admit that when this advice is carefully guarded by a number of rather important qualifications no exception can be taken to it, but the necessary reservations are such as to greatly diminish the number of cases in which the average veterinary practitioner can with safety rely upon his own microscopic skill in diagnosing anthrax.

It is obvious that in discussing this matter much depends on what is meant by an expert microscopist. When it is the diagnosis of anthrax that is under consideration it may fairly be said that the only expert is the person who has had a large experience in the examination of the blood of animals suspected of having died of anthrax. Everyone who has had such an experience is, from the present point of view, an expert, although he may not have made frequent use of the microscope in the diagnosis of other diseases. A truth that is perhaps not quite so obvious, but one which we could support by some very convincing examples, is that a large experience in the use of the microscope, and the possession of great skill in its application to the diagnosis of other diseases than anthrax, is quite compatible with a dangerous liability to error in the diagnosis of anthrax. It follows from this definition that in this country, where anthrax is, fortunately, not a common disease, very few veterinary practices can furnish, even within a long period of years, the opportunities which are necessary to qualify a man as an expert in the diagnosis of anthrax.

What, then, are the qualifications with which the advice to non-experts to rely on microscopic examination of the blood must be hedged about, if frequent errors are to be avoided in the diagnosis of anthrax? The first is, that the entirely inexperienced ought not to base their diagnosis mainly, or indeed in any degree, on microscopic examination of the blood. The second is, that unless the practitioner is provided with a good modern microscope, and has had considerable experience in its use, he will do well to employ it only in those cases of suspected anthrax in which the presence of putrefactive bacteria in the blood to be examined is impossible—in other words, he should trust nothing to microscopic examination if the animal has been more than a few hours dead.

Mr Reeks thinks that with regard to the diagnosis of anthrax the expert has been carrying too high a hand, but we are inclined to think that if experts were at liberty to disclose all the errors in diagnosis that come under their notice the plea for a more frequent use of the microscope by the non-expert would be the reverse of

strengthened. The fact is, that in the literature on the subject the difficulties which lie in the way of the inexperienced have been as a rule minimised rather than exaggerated. The simplest case of all is that in which it is known that the animal has not been dead for more than an hour or two, and in which it is therefore perfectly safe to assume that any bacteria which are found in the blood of the peripheral parts of the body must have been there while the animal was still alive. Since there is no other disease of the domesticated animals in which the perfectly fresh blood contains bacilli resembling those of anthrax in form and size, it might at first sight appear impossible for anyone to fall into error in such a case. Unfortunately, experience has shown that error is possible even in these circumstances. With an inadequate instrument the inexperienced observer may suppose that he sees bacilli where none are present, and he may fail to see them when they would be conspicuously visible to the experienced eye.

It need hardly be said that the chances of error are infinitely greater when, as is often the case, it is not known precisely when the animal died, and pronounced signs of putrefaction are present in the carcase before the veterinary surgeon is called in. In such a case the discovery of bacilli in the blood gives no assistance in diagnosis except to those who by considerable experience have acquired the ability to recognise anthrax bacilli when they occur side by side with putrefactive bacteria that approach them in size and shape.

We observe that among the disadvantages of consulting an expert Mr Reeks includes the chance—in his opinion not a very remote one—that even the expert may give a false verdict. We confess that to us this hardly appears to be the way to encourage the tyro to have recourse to the use of the microscope in the diagnosis of anthrax. If there are such pit-falls in the way of the expert, how precarious must be the path of the inexperienced! Besides, surely Mr Reeks in this matter has scarcely done the expert justice. It is perfectly true that in completely putrid anthrax blood even an expert may not be able to find any anthrax bacilli, but it is obviously wrong to say that in that event the expert “is compelled from the very nature of the evidence he obtains to return the case as not anthrax.” It is precisely the conclusion at which the non-expert is likely to arrive in such a case, whereas the expert is much more likely to know the limitations of his powers of diagnosis when dealing with putrid blood, and to decline to give any opinion in the circumstances.

After all, however, there is perhaps not much difference between the position which Mr Reeks takes up on this question and the one which we consider the safest. Mr Reeks advises the newly qualified veterinary surgeon to avail himself of the expert's assistance during his first few years in practice, and at the same time to utilise the means at his disposal for acquiring some measure of the expert's

knowledge. The plan here recommended is excellent, but we should not care to encourage the country veterinarian to discard the assistance of the expert by telling him that he is not likely to commit many serious errors by relying on his own skill with the microscope. In the interests of accurate diagnosis it would perhaps be better to warn him that for a very considerable period he will be exposed to the risk of giving a wrong opinion, and to advise him not to give his verdict too freely on the result of his own microscopic examinations. Until he has honestly satisfied himself by numerous trials that he can always recognise the anthrax bacillus when it is before him, and that he is in no danger of mistaking other organisms for it, it will be well for him to send an ear or a foot from all doubtful cases to someone in whose opinion he can place confidence.

FOOT-AND-MOUTH DISEASE.

The stock-owners of Great Britain have much reason to congratulate themselves that this disease, notwithstanding that it obtained a footing in the country in the early part of last year, has not yet assumed serious dimensions; but, on the other hand, the fact that it cannot yet be regarded as stamped out gives ground for some uneasiness. Ten outbreaks have already been notified for the current year, and, although it is now some weeks since the last of these was discovered, the experience of last year warns us not to be too ready to assume that the invasion is at an end.

In our last issue we commented on the fact that the investigations of the Board of Agriculture had failed to throw any light on the source of the infection in the first outbreak of last year, and that a similar mystery enshrouded most of the succeeding outbreaks. Our principal reason for returning to the subject is to point out a rather striking analogy between the incidence of the outbreaks since the present invasion began, and that which was observed a few years ago in another country, which also has, in this connection, the advantage of being isolated by the sea. We refer to Denmark.

It appears that from 1878 till 1892 Denmark was entirely free from foot-and-mouth disease, but it reappeared in the country in the autumn of the last mentioned year, and during the following twelve months about 400 separate outbreaks were detected. The source of the first of these outbreaks, and of many of the succeeding ones, was not ascertained. At that time the disease was very prevalent in Germany, but the importation of German cattle was prohibited. German cattle-dealers occasionally visited Denmark, and it was suspected that they were sometimes instrumental in introducing the disease, but in no case could this be definitely brought home to them.

There does not appear to be any reason to believe that any

considerable proportion of these 400 outbreaks were due to importation of the infection from Germany or elsewhere, for since at that time stamping out measures were as a rule not put into force it is natural to suppose that the virus was often carried from one Danish farm to another. In a good many cases the disease appeared to be spread by the agency of the public creameries, but it also continued to crop up in comparatively remote parts of the country, and in circumstances which afforded no clue to its origin. It was suspected that railway trucks played a part in the dissemination of the infection, and when, at Dr Bang's suggestion, regulations providing for thorough disinfection of these were enforced the result appeared to be beneficial.

An interesting feature in connection with the occurrence of the disease in Denmark during the winter 1893-94 was its reappearance on four large farms on which outbreaks had taken place from six to twelve months previously. In each of these cases it was observed that only the calves and the cattle which had been bought since the previous outbreak were attacked, the other animals being apparently immune in consequence of the first attack. In these cases an attempt to stamp out the disease was made by killing all the new cattle, but in spite of this a third outbreak occurred on one of the farms twelve months after the second. Again only the new animals were attacked, showing that the survivors from the first outbreak were still immune after two years.

Since the spring of 1896 one or two outbreaks have been annually detected in Denmark, in spite of the continued prohibition of importation of cattle from abroad, but each of these incipient invasions has been arrested by the prompt slaughter of all the animals on the affected premises. This policy was remorselessly carried out, even on very large farms and where only a single animal was as yet attacked.

In none of these recent outbreaks was it possible to trace the source of the contagion, but a striking fact in connection with them was that almost the whole of them occurred near the coast exactly opposite North Germany.

The fact that repeated outbreaks of foot-and-mouth disease have within recent years occurred in Denmark in spite of its insular position, and notwithstanding the exclusion of foreign cattle, does not appear to have attracted any attention in this country. Had it done so there would probably have been less surprise when the disease suddenly appeared in the eastern counties in the early part of last year. The history of the disease in Denmark and our own more recent experience have destroyed the sense of security which had been growing among us since the last outbreak in 1894, and they indicate that, unless there is some source of infection against which stricter precautions can be taken than those hitherto employed, we

may reasonably expect visitations of foot-and-mouth disease whenever it happens to be very prevalent near the coast-line of the Continent opposite our own shores.

In the absence of any direct evidence as to the precise manner in which the virus of the disease is introduced in these mysterious outbreaks, speculation on the point is natural. Probably the one that occurs most readily to the lay mind is that the infection is air-borne, but that may be dismissed as in the highest degree improbable, if not altogether inconceivable. There is, it is true, some evidence to show that infective material may be carried for short distances in the atmosphere without losing its vitality, as, for example, the case cited by a German veterinary surgeon¹ in which the disease broke out in a district traversed by a road running east and west, and spread from farm to farm on the western side of the premises first affected as long as the wind was in the east, and then extended in the opposite direction when the wind changed to the west. Examples of that sort, however, cannot be accepted as convincing, owing to the difficulty of excluding other possible methods of infection; and experience both at home and abroad has shown that when the agency of human beings and other living things as carriers of the contagion is excluded the disease does not spread to other animals within even a short distance. Besides, if it were possible for the virus to be carried across the German Ocean in the air, outbreaks along our eastern sea-board could hardly fail to be of very frequent occurrence at the present time, and we should not have had a period of five years of immunity from the disease.

The suggestion that birds may be the carriers of the contagion from the Continent of Europe to our own shores is by no means so far-fetched. The degree of its probability must depend mainly upon the frequency with which sea-fowl are in the habit of crossing from shore to shore in one single flight, for it is not at all likely that much danger would attach to a bird which in crossing had spent some time in the water. It is not forgotten that swallows and other migratory land birds periodically cross the ocean in one continuous flight, but the agency of any of these can hardly be called in to account for the outbreaks which began the present series in January of last year.

But of all the possible means by which the infection can be introduced from abroad at the present time, human agency appears to be the most probable. It is recognised on all hands that a person who has recently been near an affected animal, or in contaminated premises, is capable of spreading the infection, and it is also true, though perhaps not so generally recognised, that the virus may by simple contact be transferred from such a person to another, who may thus become the innocent and unconscious agent in starting an outbreak.

¹ Bericht ueber das Veterinärwesen im Königreiche Sachsen für das Jahr 1899.

Some interesting examples of this indirect spread of the contagion are mentioned in the report regarding the occurrence of foot-and-mouth disease in Saxony in the year 1899, from which we have already quoted. In one outbreak the introduction of the disease appeared to have been brought about by intercourse between the girl who attended to the animals and the wife of a man who was engaged at a farm where foot-and-mouth disease existed. In another case a cattleman carried the infection to the animals under his charge in consequence of his having sat for a short time in a shop beside the servant from another farm on which the disease had previously broken out. In a third case the first animals attacked were the cows milked by one particular milkmaid, and it was ascertained that some days previously she had been in company with a servant from an infected farm. So convinced were some of the local authorities in Saxony of the possibility of the disease being spread in this way that the children from infected farms were not allowed to attend school, and in some cases restrictions were placed on the movements of all the persons residing at places where outbreaks had occurred.

If the contagium is as easily carried as these cases would appear to show, it is in no way surprising that the disease should be occasionally introduced from abroad by travellers who have been in districts in which it is prevalent, and it is obvious that no efficient precautions can be taken against such a risk. Finally, a correct appreciation of the many subtle ways in which the agents of infection may be carried suggests that it would be very unwise to attempt to deal with any of the outbreaks occurring in this country at the present time by any other plan than the wholesale slaughter of all the animals on the affected premises.

THE TUBERCULIN TEST.

The present occasion appears to be opportune for taking stock of the position with regard to the practical applications of tuberculin as an aid to the diagnosis of tuberculosis in the lower animals, and especially in cattle. It is true that so much has already been written regarding the tuberculin test that any further contribution to the subject must be largely of the nature of a repetition; but our main motive in reverting to the question is to comment upon some rather important additions that have recently been made to our knowledge by two independent, though almost simultaneous, sets of experiments.

The first of these is the series of experiments with regard to the reliability of the tuberculin test which was carried out by the Royal Agricultural Society of England. These experiments were begun in

the summer of 1899, and completed in July last, and the report regarding them appeared in the last number of this journal (p. 368). For the most part the experiments consisted in infecting presumably healthy cattle with tuberculosis, and afterwards testing them at intervals with tuberculin in order to ascertain how soon, if at all, they would react. It was hoped in this way to add to the already existing knowledge regarding the general question of the accuracy of tuberculin as a diagnostic agent, and also to obtain information concerning a point which at that time was very obscure, viz., the period which elapses between infection and the earliest moment at which an injection of tuberculin will provoke a reaction. At present we desire to call attention only to the information with regard to this latter point which the results of the experiments provide. In this respect the results were somewhat remarkable, or at least different from what most people would probably have expected. Briefly stated, the experiments indicated that with different methods of infection, and even when animals are infected in the same way, the period which elapses before a distinct reaction to tuberculin can be obtained is very variable, and may be anything from eight days to over seven weeks. The actual periods observed were from eight to twenty-three days when the animals were infected by intravenous injection, from eight to fifteen days with subcutaneous inoculation, and from eight to fifty-one days when the infective material was administered by the mouth. It ought to be observed that it is possible, though it cannot be said that it is at all probable, that what for the sake of brevity may be called the period of incubation, may have been modified by the preliminary test with tuberculin to which the animals had been subjected in order to ascertain whether they were the subjects of naturally contracted disease or not, and by the repeated injections which were made after infection to discover the earliest moment at which a reaction could be obtained. This possible source of error could only have been avoided by using a very much larger number of animals for experiment, so as to permit of proportionally longer intervals between the tests. As already said, however, it does not appear to be at all probable that the injection of an ordinary dose of tuberculin, even when several times repeated, has any effect in preventing an ulterior reaction when it does not itself provoke a reaction. In some cases the animals were tested once a week after infection, but in the later experiments the interval between the injections was purposely made longer.

The second series of experiments throwing light on the length of the period which elapses between infection and the earliest moment at which the animal acquires the power of reacting to tuberculin, was carried out in France during the past year.¹ In that series several different methods of infection were employed. To four animals the

¹ See page 80 of this number.

infective material was administered by the mouth ; two were made to inhale air in which tuberculous material was suspended as a dry powder or dust ; two were similarly infected by distributing in the air which they breathed a fine spray of water to which tubercle bacilli had been added ; two cows had the tuberculous material introduced into the udder through the teat ; and in two other experiments the animals were respectively infected by intra-venous and intra-tracheal injection.

As in the English experiments, the date at which the first reaction was obtained varied a good deal in different cases. In the conclusions placed at the end of the report the period of incubation is said to have varied from thirty-two to forty-eight days in the animals infected by way of the mouth, and from nineteen to thirty-two days in those infected through inhalation. In this statement, however, there appears to be a slight inconsistency, for in one of the animals infected by way of the respiratory passages the temperature rose to 39.5° during the test carried out on the thirteenth day ; and in one of the two animals infected by feeding, and credited with a reaction on the thirty-second day, the highest temperature recorded was precisely the same, and in the other it was only one decimal point higher. The fact is that in none of these cases was the rise of temperature very decisive, and the only distinct reaction obtained from the animals infected by way of the alimentary canal was that which occurred on the forty-fourth day in one of them. In the animal infected by the intra-venous method, and in one of the cows infected through the teat, the temperature soon became so unsteady as to make the results of the tests with tuberculin uncertain. The first reaction from the other cow infected through the teat was obtained on the thirteenth day, and the animal infected by intra-tracheal inoculation reacted for the first time on the twenty-fifth day.

The results of these experiments are important from two different points of view. The one which may be noticed first furnished the principal motive for the French experiments. It appears that in 1899 the French Senate passed a new project of law, the purpose of which is to give the purchaser of an animal the right to return it, or to have the bargain declared void, if it can be shown that the animal in question was affected with tuberculosis at the time of sale. Any claim under this law would have to be made within thirty days after purchase, and, naturally, doubts appear to have been entertained as to whether a reaction to tuberculin, or even the demonstrable existence of tuberculous lesions, thirty days after purchase, would constitute satisfactory proof that the animal in question had been diseased at the time of sale. The Committee who carried out the French experiments appear to have interpreted the results which they obtained as evidence that this law would not be likely to involve injustice to sellers, for one of the conclusions which they draw is, that "if a cow recently pur-

chased reacts to tuberculin within the thirty days following the sale, the veterinary surgeon who carries out the test is fully justified in concluding that, *in all probability*, the cow in question was infected before sale." The qualified form of this conclusion is worthy of note, and it ought to be stated that the italics occur in the original report of the Committee. Probably a good many people will think that the conclusion, even with the qualification "*in all probability*," goes further than the results of the experiments warranted. It is admitted that in one of the experiments an animal reacted as early as nineteen days after infection, and, as we have already pointed out, another animal had a slight reaction on the thirteenth day, but this difficulty is disposed of by concluding that "in the conditions of natural contagion the period of incubation would certainly be considerably longer." It appears to us that the qualifying phrase "*in all probability*" might with advantage have been introduced into this conclusion also.

In this connection attention may be called to Experiment 17 in the series carried out by the Royal Agricultural Society's Committee. In that case the animal was infected by way of the mouth, and it had a pronounced and typical reaction when tested on the eighth day afterwards. It also reacted distinctly to no fewer than six subsequent tests, and the *post-mortem* examination revealed in the intestine lesions which could reasonably be ascribed to the experimental administration of infective material, and none there or elsewhere that appeared to have been in existence for longer than the duration of the experiment. In another experiment of the same series (No. 23) there was a pronounced and typical reaction when the animal was tested with tuberculin on the twenty-fourth day after the infective material had been administered to it by the mouth.

It, therefore, appears to be pretty well established that an animal which has been experimentally infected with tuberculosis, by a method which closely imitates one of the ways of natural infection, may react to tuberculin well within a month afterwards. In saying this the fact is not overlooked that the quantity of infective material employed in these experiments was very large—probably much larger than ever enters the body of an animal of the ox species in a single day in natural circumstances, unless, perhaps, when a calf is fed with the unmixed milk from a tuberculous udder; but it is not at all certain, indeed, scarcely probable, that the period which elapses between actual infection and the earliest moment at which a reaction to tuberculin can be obtained is greatly influenced by the quantity of infective material administered to, or naturally taken in by, the animal. The larger the number of tubercle bacilli administered at one time the greater is the likelihood that infection will be induced, but even when very large quantities are given by the mouth only a small number of bacilli may be concerned in the actual infection. In evidence of this it may be pointed out that in spite of the usual oral character of the

experiments previously cited (Nos. 17 and 23), in respect of the quantity of infective material administered, the lesions induced were by no means extensive.

When all these points are taken into consideration, it would appear to be very doubtful whether a reaction to tuberculin thirty days after purchase justifies the veterinary surgeon who carries out the test in certifying that the animal in question was in all probability infected before the date of the sale.

The second conclusion drawn by the French Committee is, that "if the lesions are softened or calcified, whatever be their extent, and no matter how discrete or limited they may be, in future the veterinary surgeon may affirm with certainty that these lesions have been in existence for more than fifty days." We cannot help thinking that this conclusion is much too positive. There is reason to think that the date at which caseation, softening, and calcification take place varies a good deal in different cases, and it is far from certain that caseous lesions never soften in less than fifty days from their commencement, especially in young animals. Moreover, it is not impossible that calcification of tuberculous lesions may set in within fifty days after their inception.

In No. 14 of the experiments carried out by the Committee of the Royal Agricultural Society, the animal was infected by subcutaneous injection in the region of the flank, and it was killed on the sixty-third day afterwards. Not only was there softened caseous material at the point of inoculation, but the adjacent precrural lymphatic gland was in a similar condition. The latter lesion was, of course, a metastatic one, and probably it had been in existence for considerably less than sixty-three days. In another experiment of the same series (*see* p. 3 of this number) the lesion at the point of inoculation was found to be softened when the animal was killed on the thirtieth day after inoculation, and there were indications of commencing caseation in metastatic lesions which must have come into existence well within the month. We do not know of any experiment proving that calcification may occur within less than fifty days, but it seems to be a little rash to assert positively that such a thing is impossible because it has not been observed in a comparatively small series of experiments.

The other aspect of the results of these recent experiments is no less interesting. In both series it was found that a very considerable period might elapse after infection before the animal had acquired the property of reacting to tuberculin. The maximum period in the French experiments was forty-eight days, and in the English experiments fifty-one days. In three of the six animals infected by way of the alimentary canal in the English series it was forty days. The important bearing of this discovery on the practical uses of tuberculin is obvious. In the first place, it indicates that in any herd of con-

siderable size in which tuberculosis has existed for a considerable time an attempt to weed out the affected animals by the aid of the tuberculin test is very likely to fail unless the test is repeated at rather short intervals during the first year. Even in the absence of such proof as has now been provided, the probability that such was the case was on a former occasion pointed out in these columns. It must be pronounced distinctly unfortunate that for six weeks after infection an animal may pass for healthy when it is tested with tuberculin. No doubt this defect can be overcome by a more frequent application of the test, but that means a considerable increase in the trouble and expense of eradicating the disease from a herd.

It is a fairly safe prediction that the facts brought to light by the experiments to which we have referred will be not unwelcome to those who have all along industriously endeavoured to spread the notion that tuberculin is an agent of no value in the diagnosis of tuberculosis, and we may confidently expect to see the fact that the property of reacting is not acquired by an animal on the day on which tubercle bacilli enters its body put forward as a reason for entirely abandoning the tuberculin test. It need hardly be pointed out that there is neither logic nor reason in such an attitude. When the test is properly carried out its imperfections are almost entirely in the direction of failing to detect the disease, and very rarely in the way of incriminating an animal that is free from tuberculosis. At the present time the agitation against the employment of tuberculin is fostered mainly by those who are in no way anxious for the discovery of an infallible method of detecting tuberculosis in the living animals, and who, having animals for sale, sigh for the return of the days when good condition and the absence of a cough were unhesitatingly accepted by a purchaser as satisfactory proof of freedom from the disease. It deserves to be characterised as a stupid delusion on the part of the owners of tuberculous herds if they imagine that intending purchasers at fancy prices are likely to refuse the assistance which tuberculin can lend them when they desire to avoid coming into the possession of animals that are already diseased. Non-reaction, unfortunately, falls a good way short of being absolute proof of freedom from tuberculosis, but in the present state of knowledge it is by far the most conclusive evidence of that kind that is obtainable while the animal is still alive. In like manner, not every animal in which the temperature rises after an injection of tuberculin is tuberculous, but the exceptions are not sufficiently numerous to seriously weaken the weight of a reaction as evidence of infection.

Reviews.

The Essentials of Practical Bacteriology. By H. J. Curtis, B.S. and M.D. (Lond.), F.R.C.S. London: Longmans, Green, & Co., 1900.

THE student of bacteriology has for some years had the choice of several text-books dealing with the subject. The present work will not enter into competition with these, but it ought to find a ready sale as a handy laboratory guide to those who for the first time engage in practical bacteriological work. The opening chapters are devoted to a description of instruments and apparatus, and directions for the manufacture of the various culture media in common use. A few of the non-pathogenic bacteria are then described, and the remainder of the book is taken up with an account of the more important pathogenic organisms, including the parasites of ring-worm and malaria, etc. The work is freely illustrated, and, without exception, the figures are excellent. The weakest and least accurate parts of the book are those referring to the bacteriology of the lower animals. As instances of actual error, we may mention that "scirrhus cord" in horses is said to be caused by the actinomyces, and that the dog is stated to be insusceptible to anthrax. The author also in his reference to the supposed connection between membranous colitis of the human subject and swine erysipelas has apparently confused the latter disease with swine-fever.

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1. A Course in Surgical Operations for Veterinary Students and Practitioners. By W. Pfeiffer, Assistant in the Surgical Clinique of the Veterinary High School, Berlin, and W. L. Williams, V.S., Professor of Surgery in the New York State Veterinary College. London: Balliere, Tindall, & Cox, 1900.
 2. Table of Veterinary Posology and Therapeutics, Etc. By George A. Banham, F.R.C.V.S., Second Edition.

THESE two little books, both issued by the same publishers, may conveniently be noticed together, as they are of the same type. Although both of them purport to be for the practitioner as well as for the veterinary student, it may, without serious disparagement of their merits, be said that they are much more adapted to the requirements of the latter than the former. They both contain the maximum of instruction or information compressed into the minimum of space, and, though that is a method of tuition which has demerits as well as advantages, it is one that continues to find favour with students preparing for examination. Both books are very neatly got up, and the first of them has a considerable number of original and very useful illustrations.

Lehrbuch der pathologischen Anatomie der Haustiere. Von Prof. Dr Th. Kitt. Second Edition, Vol. I. Stuttgart: Ferdinand Enke, 1900.

PROFESSOR Kitt's admirable text-book of veterinary pathology appeared in 1894, and the present volume is the first instalment of the second edition. The author has taken great pains to bring the work quite up to date, and it has been to some extent enlarged, and improved by the inclusion of a number of new illustrations. Our duty with regard to it can be very briefly discharged, for it is simply necessary for us to say that it is undoubtedly the best book, in any language, on the subject with which it deals.

CLINICAL ARTICLES.

THREE CASES OF FRACTURE OF THE EXTERNAL TUBERCLE OF THE HUMERUS.

By D. PUGH, F.R.C.V.S., Sevenoaks.

CASE I.—ON 9th May 1891 I was requested by Mr B. H., Turvin's Farm, Chevening, Kent, to examine a cob that was excessively lame. This cob had been turned out with some of the farm horses in the orchard some days previously, and the following morning it was found so lame that the near fore limb was carried during progression. The owner, thinking it would probably be all right in a day or so, did nothing for the animal; but, as no improvement was manifest on the 9th, I was sent for. I found the animal standing in the field, holding its foot slightly off the ground; the leg was considerably swollen from the shoulder to the knee. Immediately over the external tubercle of the humerus was a small wound, from which oozed a bloody ichorous discharge. On manipulation the animal manifested a great deal of pain, and on attempting to flex the limb he would threaten to rear, but with the slightest movement one could hear a loud crepitation of bones. On probing the wound it was evident that I had a compound fracture to deal with. The leg was held somewhat rigidly, differing in this respect from a fracture through the shaft of the humerus. To remove the loose portions of bone, cocaine was injected into the structures in the neighbourhood of the wound, and a long incision made down to the fracture; the detached pieces of bone were then dissected out. The principal piece was found to be the external tubercle of the humerus, with a base of about three-quarters of an inch taken each way. There was nothing special to note about the treatment, the parts being simply treated as an open wound. The horse went into light work in six weeks, apparently no worse for the loss of bone.

CASE II.—A chestnut filly, two years old, running in a field with a colt of the same age, both the property of Mr W. B., Park Farm, Halstead, Kent. The symptoms in this case were similar to the first, with the exception that they were not so severe, and that a little weight was placed on the toe during progression. Chloroform was given to the animal, and the portion of bone was dissected out; it was found to be just the tubercle itself. The wound was treated like the first case, and with the same good results.

CASE III.—This case occurred in a grey hunter, the property of Alfred Laurie, Esq., J.P., Rockdale, Sevenoaks. When I asked the owner if I might use his name in connection with the case, he said, "Certainly, and I shall be delighted to show the horse to anyone interested in it." This horse broke out of his box into an adjoining

paddock, his company being immediately resented by the sole occupant, an old carriage horse, which promptly kicked him in the region of the humerus, causing a compound fracture, and the same mechanical interference to progression that was witnessed in case No. 1. This horse was cast and chloroformed, and the detached portions of bone were removed; they consisted of the external tubercle and about three inches of the external wall of the shaft of the humerus. The subsequent treatment was the same as in the two previous cases, and the animal was going sound in about six weeks, but was rested for another six weeks before being ridden. From that date (14th August 1896) to the present day the horse has never shown any lameness.

As far as I can learn, there are no previously reported cases of this particular fracture. I have therefore given the names and addresses of the respective owners of these unfortunate animals for the sake of authenticity.

CASTRATION OF COLTS BY THE LIGATURE METHOD.

By H. A. WOODRUFF, M.R.C.V.S., Royal Veterinary College,
London.

WITHOUT offering any opinion as to the most surgical method of performing this operation, it has often been somewhat of a mystery to me that hæmostasis brought about by the actual cautery or by *ecraseur* should be quite safe, whereas ligation of the vessels of the cord should (at any rate by reputation) be attended with grave risks. A communication in one of the journals from a veterinary surgeon, stating that he always adopted the method of ligation, led me to put the matter to the test.

Since January last six horses, varying from a two-year-old to an aged thoroughbred, have been castrated by this method. The animals have in each case been cast, the usual antiseptic precautions taken, and the scrotum incised in the ordinary way. In the first three cases the non-vascular part of the cord was simply cut through with a knife after applying a silk ligature to the anterior vascular portion, the ligature in each case transfixing the cord to prevent slipping. When this was done there was certainly a little dripping of blood for about ten minutes after the animal was let up. In the last three cases a double ligature was used, and it was passed through the middle of the cord, cut and tied on either side, thus ligaturing both portions of the cord. When this method was adopted there was absolutely no bleeding at all.

In all six cases there has been comparatively little swelling after the operation, and all the animals have done well.

It may be said that an opinion formed after six cases is worth little, but personally I have faith in the method, and feel quite warranted in doing more, and in recommending it to the attention of others.

HYSTERECTOMY IN THE BITCH.

By the Same.

I AM indebted to Mr F. E. Mason, M.R.C.V.S., one of the College tutors, for the following notes of two operations performed by students under Mr Mason's supervision.

CASE I.—The first subject was a pug bitch about six years old, in a very weak condition from protracted labour. The history was that she had two days previously passed a dead and partly decomposed puppy. She was some days over her time. Attempts were made to remove the remaining pups by the natural channel, but these were unsuccessful, owing to the large size of the pups for so small a bitch. Finally it was decided to remove the uterus and its contents by operation. The patient took chloroform badly, although it was cautiously administered, well diluted with air. After preparation of the operation area, an incision was made in the middle line of the abdominal wall just behind the umbilicus, a ligature was applied to the fundus of the uterus, and the latter cut through immediately in front of the ligature; then the body of the uterus was lifted out of the abdomen. The right horn was incised and the foetus withdrawn. Next a ligature was placed above each ovary, and these organs were removed, together with the whole uterus, the broad ligaments being torn. Unfortunately, in doing this, a few drops of putrid fluid from the uterus were allowed to escape into the abdominal cavity, which was therefore thoroughly rinsed out with a 1 per cent. solution of chinisol, and the vagina was injected with the same. The incision in the abdominal wall was then sutured in superposed layers, and the bitch was allowed to regain consciousness.

When seen two days later the temperature was slightly elevated, but the wound was doing well, and small doses of quinine were given. The case was seen at intervals of three weeks, and made a complete recovery.

CASE II.—The second subject was a half-bred bull and fox terrier bitch about three years old. The history was that she was over her time, labour pains had come on, but with no result, and there was an offensive discharge from the vagina. All attempts to get the pups away by the natural opening were fruitless, so it was decided to operate.

The patient was a good subject, being strong and in good condition, and she took chloroform well. The operation was proceeded with precisely as in the first case, and the peritoneal cavity and vagina were both washed out with a 1 per cent. solution of chinisol. The uterus was found to contain five dead pups in a semi putrid condition, and all fully developed. The wound healed by first intention, and the animal made a speedy and complete recovery.

The success of these two cases is probably due to thorough disinfection of wound area, instruments, etc., to the washing out of the peritoneal cavity, and to the general antiseptic precautions observed.

A CASE OF CYSTOTOMY IN THE HORSE.

By Veterinary Surgeon A. CHINNIAH, Colombo, Ceylon.

History.—A black waler gelding pony, about 14½ hands high, eight years old, was brought to my surgery with the history that the animal passed highly blood-coloured urine. It was treated by me for some time with palliative drugs, as in cases of ordinary hæmaturia, but when the case assumed an obstinate nature I suspected that there must be some mechanical agent, such as a calculus, doing the mischief. The animal was examined per rectum, and the presence of a calculus was diagnosed with certainty.

Symptoms.—There was painful straining during micturition, but at times the urine passed was of normal colour and consistency. After brisk exercise the urine passed contained blood corpuscles and pigment in abundance. The expression of the patient during the passing of urine indicated great pain. The appetite was all throughout good, and there was no noticeable rise in temperature. As soon as the proper diagnosis was arrived at, I informed the owner that the animal must undergo an operation, which, if it succeeded, was the only means of saving the animal.

Operation.—The instruments taken for the operation were scalpels, sharp-pointed bistoury, Syme's artery forceps, bull-dog forceps, stone forceps, lithotrite, a male catheter, and a female washing catheter.

The animal was thrown on the near side, and general anæsthesia (chloroform) was induced. The penis was pulled out, washed and lubricated, and the catheter was passed. The penis was retained throughout in this position by an assistant told off for the work. The off hind leg was drawn a little forward. I took my position just near the croup. The tail was washed antiseptically, and given in charge of another assistant to be kept in the desired position. An incision was made two inches below the anus, along the middle line of the perineum, downwards. When the urethra was reached the sharp point of the bistoury was introduced into the tube (urethra), which was eventually slit open to admit the passage of the forceps. When the forceps were in the bladder the stone was grasped, with the assistance of my left hand in the rectum. Several attempts were now made to remove the stone without performing cystotomy or lithotrity, but they proved futile.

Though the lithotrite was at hand ready for use, I preferred to perform cystotomy. The bistoury was passed along the urethra into the neck of the bladder, and, guided by my left hand per rectum, to prevent any rectal fistula, an incision was made dividing the neck of the bladder, prostrate glands, and (unavoidably) the pudic artery. The latter was promptly caught and ligatured. This made the operation simple, and with the help of the hand per rectum the removal of the calculus was easy enough.

The calculus, which is shown in the accompanying figure, weighs 3½ ounces, and its circumference is 7¼ inches. It is not spherical, but convex on both surfaces, and the margin is unbroken and round. The outer layer is apparently composed of phosphate of lime, and the inner of oxalate of lime.

After Treatment.—The bladder was washed out with a tepid solution of boracic acid. The wound was antiseptically treated (painted with iodoform and eucalyptus oil), and left open. The animal micturated soon after the operation, and passed urine both through the wound and along the natural passage. The passage through the wound



Cystic calculus (natural size).

ceased after the first day of the operation—27th January 1901. The wound is being daily dressed antiseptically with what I call antiseptic emulsion, composed of boracic acid, carbolic acid, iodoform, eucalyptus oil, and glycerine, and satisfactory progress continues to be made. The wound is two inches in length and a quarter of an inch deep.

AN INTERESTING BOG-SPAVIN CASE TREATED BY FREE INCISION AND NEURECTOMY.

By FRED. HOBDAV, F.R.C.V.S., London.

IN June last I was consulted by Mr E. R. Harding, M.R.C.V.S., regarding a valuable Cleveland bay mare, six years old, suffering acute lameness from the presence of an enlarged hock. The latter showed a bog-spavin and thorough-pin, making the joint quite as big as an ordinary football and very tense. The mare was in a lot of pain, and continually rested the leg.

On the 4th of June an aspirator was applied, some of the fluid being drawn away, and a solution of iodine and chinosol in water injected. Temporarily there was an improvement in appearance, but the lameness was as great as ever, and a second operation was decided upon.

On the 20th of July, under strict antiseptic precautions and with the aid of chloroform, an incision fully 3 inches in length was made

right into the true hock joint. A very large quantity of synovia, including nine or ten coagulated lumps, escaped. The interior was carefully swabbed out with chinosol lotion and boiled wadding, and plugged before the patient was allowed to get up. Digital examination of the interior and exterior of the joint now revealed enlargements which were not perceptible before.

Mr Harding personally attended to the wound afterwards, and recovery was quite uneventful and without formation of pus.

On the 18th of August the wound had completely healed, and the hock was very much reduced in size, in fact by quite one-half, but the exostoses could still be felt, and the mare was still lame.

On this date peroneal and posterior tibial neurectomies were performed, and the animal became practically sound at once.

As soon as the wound healed she was sent to work, and is still working regularly, and quite sound. The hock is now, Mr Harding informs me, not more than a quarter the size it was before the operation; in fact, the inside where the incision was made is practically normal.

Abstracts and Reports.

THE PERIOD OF INCUBATION IN TUBERCULOSIS, AND THE AGE OF TUBERCULOUS LESIONS.

At the instigation of the Société de Médecine Vétérinaire Pratique, a committee was appointed in France early last year to carry out experiments that might throw light on the period of incubation in tuberculosis in cattle, and the age of tuberculous lesions as indicated by their appearance. For this purpose a sum of 2000 francs was placed at the disposal of the committee, and the experiments were begun in May last. The general plan of the experiments was to submit the animals to a preliminary test with tuberculin, in order to ascertain that they were not already the subjects of naturally contracted tuberculosis, to then infect them in different ways with the disease, and afterwards test them at intervals of six days with tuberculin. Finally, the animals were to be killed and submitted to *post-mortem* examination. The following is condensed from the report of the experiments made by MM. Nocard and Rossignol:

May 2nd to May 13th. The animals of this lot, numbered 1, 2, 3, and 4, were submitted to gastrointestinal infection. On the 8th May an attempt was made to infect them by giving to each 475 grammes of finely divided pleural tubercles mixed with some bran. Only one of the animals, No. 3, consumed the whole of its portion; No. 2 took about 100 grammes of a tuberculous emulsion mixed with about three litres of water. On the 14th May a further attempt was made to infect Nos. 1, 2, and 4 by giving to each of them 450 grammes of tissue from a tuberculous mediastinal gland, and of other tuberculous material in the way of softening. The tuberculous tissue was finely minced, mixed with water, and administered by means of a bottle.

As in the other cases the animals were tested with tuberculin on the 14th May, and every six days afterwards. It was not until the thirty-second day

that a reaction was obtained from any of them. When tested on that day the temperature of No. 3 rose to 39.5° , and that of No. 4 to 39.6° . In No. 2 the first reaction was obtained on the forty-fourth day of the experiment, upon which occasion the temperature rose to 40° . No. 1 never reacted at all.

Nos. 3 and 4 were killed on the 9th June, and Nos. 1 and 2 on the 1st July. The *post-mortem* examination revealed tuberculous lesions in Nos. 2 and 4, but no such lesions were discovered in the other two animals.

Infection by Inhalation of Dried and Powdered Tuberculous Material.—This series comprised two cows, Nos. 5 and 6. On the 8th May they were made to inhale air in which dried and powdered tuberculous material was suspended by means of an insufflator. The operation lasted five minutes in the case of No. 5, and four minutes and a half in the case of No. 6. The first reaction from No. 5 was obtained on the 8th June, and a second decided reaction was obtained on the 14th June. There was a slight reaction on 24th June, and again a decided reaction on the 28th of the same month. No. 6 reacted slightly for the first time on the 26th May, and it again reacted distinctly on the 1st and 8th June.

Cow No. 5 was slaughtered on the 2nd July, and the *post-mortem* examination revealed the presence of a considerable number of miliary tubercles in the lung tissue. The bronchial and mediastinal glands were enlarged, and tubercles were also present in their cortex.

Cow No. 6 was killed on the 10th June. In this case also the *post-mortem* examination revealed the presence of miliary tubercles in the lungs. The bronchial glands were enlarged, but did not show any visible tubercles. Numerous tubercles, however, were present in the enlarged mediastinal glands.

Inhalation of Finely Divided Tuberculous Material in the Moist State.—This series comprised two cows, Nos. 7 and 8. They were infected on the 8th May by causing them to inhale, in the form of a fine spray, a liquid containing tubercle bacilli. The inhalation was continued for seven minutes in the case of No. 7, and for five minutes in the case of No. 8.

No. 7 had a slight reaction when tested on the 21st May, and a more decided one on the 27th May. It scarcely reacted at all to five subsequent tests. No. 8 reacted slightly on the 27th May, and decidedly on the 2nd June. It again had a slight reaction on the 9th June.

No. 7 was killed on the 2nd July, and the *post-mortem* examination revealed a considerable number of miliary or pea-sized tubercles in both lungs, and tubercles were also present in the bronchial and mediastinal glands.

No. 8 was killed on the 8th June. In this case also the *post-mortem* examination revealed miliary tubercles in the lung tissue, and tuberculous lesions in the pharyngeal, bronchial and mediastinal glands.

Intra-venous Inoculation.—On the 6th May a cow, No. 9, was infected by the intra-venous injection of 10 cc. of a tuberculous emulsion. As early as the same evening the cow showed manifest symptoms of disease, including a rise of temperature. From that date until the 22nd May the temperature was subject to oscillation, but after the 22nd it was always above normal. The condition of the animal became very bad, the temperature rising to 40.7° , and the pulsations to 100 per minute. The animal died on the 10th June, and the *post-mortem* examination revealed a considerable number of very minute tubercles in the lung tissue, and enlarged mediastinal glands, lymphatic glands, and the trachea.

Inflammatory Inoculation.—On the 10th May a cow, No. 10, was infected by injecting into the anterior tibial space 5 cc. of a tuberculous emulsion. The animal was kept in a stable with a fine soft canula, introduced through the ear, for the purpose of observing the reaction.

By the 10th May the affected quarters in No. 10 had become swollen, hot, and tender, and the milk was already altered in appearance. Subsequently, the swelling and sensitiveness of the quarters increased, and the animal rapidly lost condition. From the third day after infection the temperature was nearly always above normal.

The cow was killed on the 10th June, when it was unable to rise. The *post-mortem* examination showed that the two uninoculated quarters were healthy, but the two into which the injection had been practised were enormously enlarged and the seat of extensive tuberculous lesions. The supra-mammary lymphatic glands were five times the normal size.

In No. 11 the temperature remained normal for the first thirteen days after inoculation, but on the twenty-first day there was a reaction to tuberculin, and the inoculated quarters of the udder were, for the first time, noticed to be enlarged, hot, and painful, and the milk contained some coagula. Between the 21st and the 25th May the temperature was unsteady, but there was a distinct reaction to tuberculin on the 26th May. At that date the infected quarters had become very large, hot, and sensitive to pressure, and the milk contained coagula and muco-pus. On the 1st June there was complete suppression of the appetite, considerable emaciation, and very great prostration, and, subsequently, all the symptoms became aggravated.

The animal was killed on the 10th June, and the *post-mortem* examination revealed a state of affairs similar to that present in the preceding case.

Intra-tracheal Inoculation.—The subject of this experiment was a cow near the calving. On the 8th May 10 cc. of a tuberculous emulsion were injected into the trachea. The cow calved three days afterwards. Subsequently it continued in apparent health, with a normal temperature, and there was no reaction to tuberculin on the 14th, 20th, or 26th May, or the 1st or 8th of June, but there was a somewhat doubtful reaction on the 15th June.

The animal was killed on the 20th June. There was a small subcutaneous lesion at the seat of inoculation, and numerous small granulations in the mucous membrane of the trachea. Tuberculous lesions were also present in the pharyngeal, bronchial, and oesophageal glands, but not in the lungs.

Infection by means of Tuberculous Milk.—For this experiment the calf of the preceding cow was used. From its birth, on the 11th May, it was fed exclusively with the infected milk from Cows 10 and 11. The milk was very rich in tubercle bacilli. The temperature remained normal until the 26th June, and when tested on the following day the calf had a pronounced reaction.

When the calf was killed (apparently on the 2nd July) tuberculous lesions were found in one of the pharyngeal glands, and in two of the mesenteric glands.

Conclusions. The Committee believe that the result of these experiments justify the following conclusions.

1. Ingestion constitutes an infinitely less efficient method of infection than inhalation. In spite of the enormous quantity of tuberculous material which was ingested, one cow out of the four resisted infection. The three others became infected, but in such a feeble degree, that in one of them it was not possible to discover the lesion which had provoked the reaction to tuberculin. In the two others the tuberculous lesions were of very small extent, and they would certainly have escaped detection had the *post-mortem* examination been less minute. Even the calf, which from the day of its birth was fed on the milk furnished by Cows 10 and 11, which was found to be only slightly affected, although the milk was extremely rich in tubercle bacilli. The period of incubation varied from thirty-two to forty-eight hours.

2. Inhalation is the ordinary and most efficacious method of infection in tuberculosis. The results of the experiments were always the same, whether the tuberculous material was inhaled in the state of a dry impalpable powder,

or as fine particles of liquid holding the bacilli in suspension. The period of incubation varied from nineteen to thirty-two hours.

Direct injection into the trachea did not yield the results that might have been expected. The lungs completely escaped infection. This is because the liquids injected never reach the pulmonary alveoli; they do not get farther than the small bronchi, and, as is well known, the powers of phagocytory defence of the bronchial mucous membrane are very strong. The enormous quantities of bacilli injected were surrounded by phagocytes, and eliminated with these in the mucus of expectoration.

Even in the cows infected by inhalation of the moist powder the bronchi, bronchioles, and pulmonary alveoli escaped infection. The tubercles were situated under the pleura, or at the periphery of the lobules, in the interstitial cellular tissue. It is probable that each focus of disease was formed around a phagocyte of the bronchial mucous membrane, which had re-entered the lymphatic circulation after having taken in one or several tubercle bacilli.

3. Unlike the bronchial mucous membrane, the mucous membrane of the excretory canals of the active mammary gland offers a feeble defence against bacterial infections, and notably against the bacillus of Koch. These experiments, and others of a similar kind which have been carried out on milch goats, show that of all the tissues of the living body the active mammary gland constitutes the most favourable medium of culture for the tubercle bacillus. They also show the possibility, which some authors have denied, of a primary tuberculosis of the mammary gland.

Finally, they show that there is in reality such a thing as tuberculous intoxication, for Cows 10 and 11, although they were at the point of death when they were killed, had no organic lesion capable of explaining the profound cachexia from which they were about to succumb. The period of incubation was very short in these two cases—viz., three days for No. 10 and thirteen days for No. 11.

4. As is always the case when one operates with a virulent bacillus, intravenous inoculation proved to be the most severe and most rapid method of infection. However, one cannot draw from it any useful conclusion, as it is never a method of natural infection.

5. Finally—and this observation is highly important from the practical point of view—in none of the experimental animals had the lesions undergone the softening or calcification which may be said to be the rule in bovine tuberculosis.

As regards the two principal points which the Society had in view, the results of the experiments are summed up as follows:—

1. Whatever be the method of infection, a certain interval elapses between the moment when the contagium penetrates into the body and that at which it manifests its effects by a reaction to tuberculin. The duration of this interval is variable. In these experiments, in which the chances of infection were at a maximum, it was from nineteen to thirty-two days in infection by inhalation, and from thirty-two to forty-eight days in infection by ingestion. It is certain that in the conditions of natural contagion the duration of the period of incubation is notably longer.

It follows that if a newly bought cow reacts to tuberculin within thirty days after purchase, the veterinary surgeon who carries out the test is fully justified in concluding that, in all probability, the cow in question was infected before the date of sale.

2. One often finds it difficult to determine the age of tuberculous lesions found at the autopsy, and the experiments carried out by the present author show that, if the lesions are situated in the lungs, whatever be their extent, and however discrete and small they may be, the veterinary surgeon will henceforth be able to affirm with certainty that such lesions have been in existence for more than fifty days.

THE INSPECTION OF MEAT AND MILK IN GLASGOW.

FORMERLY the inspection of meat in Glasgow was under the dual control of the Chief Constable and the Sanitary Inspector, but at the end of the last financial year it was placed under the supervision of the veterinary surgeon to the Corporation, Mr A. M. Trotter. At that time an assistant veterinary surgeon and seven meat inspectors were appointed, but this number was soon found to be quite inadequate, and the staff has since been augmented by the addition of two assistant veterinary surgeons and seven meat inspectors.

The following is extracted from Mr Trotter's Annual Report to the Corporation.

INSPECTION OF MEAT.

From the 1st June to 31st December there were slaughtered:—

<i>Cattle.</i>	<i>Calves.</i>	<i>Sheep.</i>	<i>Pigs.</i>	<i>Goats.</i>
58,375	1,244	184,588	31,882	15
of these—				
<i>Cattle.</i>	<i>Calves.</i>	<i>Sheep.</i>	<i>Pigs.</i>	<i>Goats.</i>
1,368	—	158	524	—

were partially or totally condemned.

Tuberculosis.—The most important disease, considered from a hygienic as well as from an economic standpoint, was tuberculosis. Of the 23,587 home cattle—oxen, bulls, cows, heifers—dressed in Moore Street, Victoria, and Milton Abattoirs, the serous membrane of the pleural or peritoneal cavities of 1035 or 4·38 per cent. were affected; 328 or 1·39 per cent. were partially, and 707 or 2·99 per cent. were totally destroyed, as unfit for human food. These figures, although showing the great prevalence of this disease among home cattle, do not, however, give any indication of the extent of the ravages, as in the majority of animals the lesions are confined to the viscera.

Of the 31,882 home swine, 481 or 1·58 per cent. were affected, 46 or ·14 per cent. were totally, and 435 or 1·36 per cent. were partially condemned. The disease was found localised in 435 swine in the lymphatic glands of the throat, and consequently the head only was destroyed.

During the period, 34,788 cattle arrived at Yorkhill, and an examination of the viscera after slaughter showed that 25 or ·07 per cent. were more or less affected. Of these, 3 (·008 per cent.) were totally destroyed, and 5 (·014 per cent.) were partially destroyed, whilst in 17 (·048 per cent.) the internal organs only were condemned.

Anthrax. Three cases of anthrax were discovered, and were reported in terms of the Diseases of Animals Act, 1894.

In one instance the dressed carcass of a bull was sent for examination prior to being delivered to the meat salesman in the Corporation Dead Meat Market. On the previous evening the animal appeared in good health, but on the following morning the attendant found it in a dying condition. It was at once bled, and, as it was believed to have choked on a turnip, the carcass was considered by the farmer to be fit for human food. In this instance the principal organs were sent in along with the carcass. The other cases were those of animals that had died in the vicinity of the city, and had been brought in for the purpose of being dressed.

Carcinoma.—The carcasses of five cows were objected to on account of the

From 1st June to the 31st December there were examined at the various railway stations, wharves, etc., the following:—

<i>Beef.</i>			<i>Veal.</i>		<i>Pork.</i>		<i>Mutton.</i>	
<i>Sides.</i>	<i>Qrs.</i>	<i>Cuts.</i>	<i>Car.</i>	<i>Cuts.</i>	<i>Car.</i>	<i>Cuts.</i>	<i>Car.</i>	<i>Cuts.</i>
810	1274	158	4389	48	897	496	2451	7

Stations.—From 1st June to 31st December 2449 visits were made to stations. Of the meat examined, twenty-five tons, comprising, of the aggregate weight of 4337 lbs., were found to be safe for human food. These include five carcasses of beef, seventeen of veal, and one of pork. On every day it was ascertained that the consignments in the markets were made safe, and consequently legal proceedings were avoided against the vendors. In one case a party in Armadale was fined £10 for selling butter from a barrel. On examination it was found to be contaminated with the bacteria *Bacterium coli*, having been tampered with by a woman who had purchased it there. The charge was found proven and she received thirty days' imprisonment. The woman was married and lived with her farmer in Denny. On another occasion a cow which had been found dead in the byre, but not reported by the owner as being unfit for consumption for human food. The magistrates ordered the carcass to be buried, with the alternative of turning it into manure.

Wharres.—During the

various wharves, with the result that four carcasses of beef and five of pork were dealt with. The cause of seizure was tuberculosis and decomposition, and entailed the destruction of 1536 lbs.

Shops.—7153 butchers' shops were visited, and in 269 instances meat was seized and destroyed. These seizures comprised 1897 lbs. of beef, 235 lbs. veal, 350 lbs. mutton, 57 lbs. pork, and 144 lbs. sundries—in all, 2683 lbs. Legal proceedings were instigated in one case, when the accused was found guilty and fined £3 3s., with the alternative of fifteen days' imprisonment.

Sausage Factories.—124 visits were made to sausage factories, but nothing of an objectionable nature was detected.

Hide Markets.—At these places an inspector calls every morning, but calves are brought in at all hours, and it is simply impossible for the department to examine all arrivals without having an inspector constantly on duty. Consequently a large number is permitted to pass into consumpt without being inspected. During the period 380 visits were made, and 47 calves were seized and destroyed.

Dead Meat Markets.—The dead meat markets were visited several times a day, and the meat consignments examined. The majority of the meat salesmen have cordially assisted the inspectors by acquainting them of country consignments arriving between their visits. There were seized and destroyed with the owners' consent:—

<i>Beef.</i>			<i>Veal.</i>		<i>Pork.</i>		<i>Mutton.</i>	
<i>Sides.</i>	<i>Qrs.</i>	<i>Cuts.</i>	<i>Car.</i>	<i>Cuts.</i>	<i>Car.</i>	<i>Cuts.</i>	<i>Car.</i>	<i>Cuts.</i>
14	60	4	23	—	2	7	—	10

Miscellaneous—four boxes pig kidneys.

Examination Station.—The difficulty of thoroughly and efficiently examining meat consignments at the stations, wharves, etc., caused Mr Trotter to approach several of the large consignees of pork with the view of having all meat consigned to them brought to Moore Street abattoir for examination, prior to it being taken to their place of business. These gentlemen promptly acquiesced, and their action has been followed by others, and now at least 75 per cent. of all imported pork is brought for examination before being offered for sale for human food. The importance of this departure cannot be overestimated, and, more especially, when it is remembered that these firms do this voluntarily, and at considerable expense and trouble.

Prior to this time pork was exempt, or nearly so, from inspection, but the necessity of its being examined is demonstrated by the experience of the past three months. During this time, 347 swine were brought for examination, and, of these, 12 or 3·4 per cent. were found to be affected with tuberculosis. The importance of an examination station will be readily understood, when it is stated that pigs are consigned either in large hampers containing six or nine carcasses, or each carcase is sewn in canvas, rendering it impossible to examine them in transit. The necessity of places where imported meat can be thoroughly examined, with the minimum amount of handling, is recognised in all Continental countries, where these stations are recognised as indispensable for an efficient system of meat inspection.

Disposal of Unsound Meat.—All diseased meat was hitherto placed in an aqueous solution of picric acid and sulphate of iron for at least several hours. The intention was to render the meat so offensive to the senses that it would be impossible to use it for human food. Meat so treated acquired a greenish-yellow colour. It was found, however, that the picric acid did not penetrate more than one-sixteenth of an inch, and, therefore, the removal of the surface layer was sufficient to obliterate all traces of dipping. The tallow obtained from meat so treated was stated to be deteriorated in value.

The following experiments were undertaken to ascertain the effects on meat of crude carbolic acid, cresylic acid, picric acid, heavy oil of tar, and chloride of lime :—

Series 1.

Several large portions of meat were freely sprinkled with :—Crude carbolic acid, cresylic acid, and heavy oil of tar.

After having lain for twenty-four hours the surface layer was removed, and the tissue underneath was found to be normal.

The fat obtained from the meat, after prolonged boiling, smelt of the chemical used.

Series 2.

From one animal a small quantity of fat was taken, and divided equally among three flasks. To these were added respectively—(1) crude carbolic acid, (2) chloride of lime in solution, (3) picric acid.

After being exposed to the action of steam for four and-a-half hours, they were compared with fat taken from the same animal, which had been treated in a similar manner, but without the addition of any chemical.

The results were as follows :—

<i>Chemical.</i>	<i>Colour of Fat.</i>	<i>Smell.</i>	<i>Consistency.</i>
Carbolic acid.	Not materially altered.	Slight.	Not materially altered.
Chloride of lime.	Bleached.	Do.	Do.
Picric acid.	Yellow.	Do.	Do.

Series 3 (a).

A solution of crude carbolic acid in the proportion of 1 part in 1000 of water was made up, and a carcase cut up in the usual way for dipping was permitted to remain in it for thirty-six hours. On removal the surface of the carcase had a bleached look, but on section, it was found that the discolourisation had only penetrated to the extent of half an inch. The tissue underneath the surface layer was normal in colour, taste, smell, and touch. The surface of the carcase which had been exposed to the solution emitted a strong odour of carbolic acid, but, owing to the unsatisfactory penetrating power of the solution, it was deemed unnecessary to proceed further with the experiment.

Series 3 (b).

Into a strong solution of the chloride of lime a piece of meat was submerged for twenty-four hours. At the expiry of that time the surface of the muscular tissue was found to have acquired a dirty greenish-yellow colour. The penetrating power of the solution may be said to be *nil*, as a protecting layer is formed on the surface of the meat.

These experiments were continued on a commercial scale, through the courtesy of Messrs Couper & Bryce and Mr A. S. Bryce, who placed at the Chief Inspector's disposal large quantities of diseased meat. The result demonstrated the unsuitability of these agents for attaining the object desired.

The committee, after due consideration, decided to have all unsound meat removed from the various slaughter-houses to the works of the purchasers in a covered cart. The covers of these carts are locked and sealed by an inspector before leaving the slaughter-house, and cannot be opened in transit. The various firms purchasing the meat are required to give a guarantee that all meat purchased by them will only be used for boiling down purposes, and that the cart will be taken by a route mutually agreed upon.

INSPECTION OF MILCH COWS.

During the year 1220 visits were made to cow-sheds, and 13,919 milch cows were examined. The milk of fifty cows was temporarily or permanently withdrawn from circulation; nine permanently on account of tuberculosis, and eight for various other diseases.

During the past few years the standard of dairy stock has much improved, and at present compares favourably with those of other parts. The majority of the animals are in their prime for dairy purposes, yielding not only a large quantity, but milk of superior quality. Nearly all are kept for one period of lactation only, consequently the average life of cows in our city byres is about nine months. If the animals are brought into the cow-sheds in a healthy condition, their sojourn is so short that they cannot become infected with tuberculosis to any great extent.

Several cow-feeders are in the habit of purchasing Irish cows in order to minimise the risk of adding tubercular animals to their herds. It is well known that bovine tuberculosis is almost unknown in certain districts in Ireland. This is undoubtedly due to the cattle leading a natural existence in the open air.

Inspection of Milch Cows in Country Districts.—The inspection of the dairy stocks in the country can only be overtaken during the winter months, when the animals are housed during the greater part of the day. In the early part of the year twelve farms in Ayrshire were visited, and 304 cows were examined. In one instance the udder was found to be tubercular, and the animal, under the powers of the Glasgow Police (Amendment) Act, 1890, was removed from the premises.

Milk for Corporation Fever Hospitals.—Forty-three visits were made to the three farms supplying milk to the Corporation fever hospitals, and in three instances cows were detected suffering from affections rendering the milk unsuitable for human consumption. The cows on these farms are guaranteed free from tuberculosis by passing the tuberculin test. The test was applied by veterinary practitioners, and on the strength of their certificates sixty-seven animals were permitted to enter the herds.

Inspection of Cattle under The Public Health (Scotland) Act, Section 43.—Under this section the cattle exposed for sale in the Corporation cattle market were examined, and four animals were destroyed with the consent of the owners. On one occasion a cow was seized, and as the circumstances of the case were such as to warrant prosecution the case was taken into Court, and the accused fined £5 with the alternative of fourteen days' imprisonment.

THE CONDITION OF THE AIR OF BYRES.

An investigation was undertaken by Mr Trotter to determine the minimum amount of pollution present in the air of byres, and he adopted Pettenkoffer's bottle method as being the most accurate and suitable for the purpose. A jar of about five litre capacity was filled with water, and emptied at a height of about eighteen inches from the ground. 100 cc. of baryta water was then added, and, after being agitated at frequent intervals for an hour, it was decanted into a small stoppered bottle and set aside to permit the soluble barium carbonate to separate out. A known quantity of the clear supernatant fluid was then titrated with a standard solution of oxalic acid, with phenol phthaleine as indicator, and from the diminished alkalinity of the original baryta water the quantity of carbonic acid present in the air was calculated. A wet and dry bulb thermometer was suspended in the byre near the place where the sample of air was collected, and another in the courtyard. Several readings were taken to ensure accuracy before being recorded. The results are thus summarised—

Cubic feet per Animal.	Floor area per Animal.	Amount of Carbonic Acid.	TEMPERATURE.				Dewpoint.		Relative Humidity.	
			Inside. Dry Bulb.	Wet Bulb.	Outside. Dry Bulb.	Wet Bulb.	Inside.	Outside.	Inside.	Outside.
The carbonic acid was less than 10 in 10,000 in 7 cases.										
775.50	52.27	8.81	54	50.7	43.64	41.28	47.43	38.23	77.7	81.5
The carbonic acid was between 10 and 15 in 10,000 in 21 cases.										
691.19	59.50	12.17	53.71	50.51	35.97	34.23	47.36	32.03	79.30	72.38
The carbonic acid was between 15 and 20 in 10,000 in 15 cases.										
844	72.34	17.18	58.1	54.4	41.07	39.59	51.06	39.06	77.60	—
The carbonic acid was between 20 and 25 in 10,000 in 1 case.										
985.43	80.11	20.38	50	48	34.5	33	45.83	30.48	85.5	85.4
The carbonic acid was between 25 and 30 in 10,000 in 3 cases.										
614.40	41.27	26.38	61.8	59.3	42.9	39.8	57.27	36.27	85.78	79.4
The carbonic acid was over 30 in 10,000 in 1 case.										
651	64.25	33.13	62	60	43	41	58.28	38.60	87.4	84.4

The augmentation of carbonic acid is due to (1) the interference with the free movement of the air, (2) the increased amount of fermentation and decomposition of organic matter, (3) the greater combustion of fuel, (4) the greater activity of life, and (5) the absence of vegetation.

The diffusive power of gases is by itself incapable of maintaining a uniform composition of the aerial sea, but requires the aid of the more powerful agencies, currents, storms, rains, etc.

The proximity of a dungstead is a grave source of air pollution. For instance, No. 31 was a comparatively new byre, in which the doors, windows, and ventilating openings are all placed in the wall facing a large dungstead, situated some 12 feet distant. In a sample of air collected midway, with the wind blowing over the dungstead in the direction of the byre, carbonic acid was found in the proportion of 7.98 parts per 10,000. The influence of the deleterious gases on the health of the lower animals is unfortunately not universally recognised, and, as the aim is to minimise labour, the dungstead is placed as near the byre as possible; indeed, it is frequently found underneath the same roof, and communicating by means of an opening. The gases, liberated by the fermentation and decomposition of the accumulated manure, ventilate into the byre through the opening, and in a less degree, by the process of diffusion, which is constantly going on through the walls themselves. Consequently, the air of the cowshed next to the dungstead contains a much larger amount of carbonic acid. Two experiments bearing on this point were carried out with the following results:—

No.	Parts of CO ₂ per 10,000		
	End next Dungstead.	End away from Dungstead.	Increase.
1.	14.6	13.34	1.26
2.	13.37	11.95	1.42

It has been demonstrated that the air of a building which has been closed for some time is richer in carbonic acid than that of the surrounding atmosphere. This augmentation is due to the presence of dirt; every particle of which is constantly adding its quota of deleterious gases. It stands to reason, therefore, that the proper domain of ventilation begins when cleanliness has done its best. The influence of filth as a source of air pollution cannot be over-estimated, but its importance could be materially diminished by scrupulous cleanliness.

During respiration, atmospheric air undergoes marked changes: the temperature is raised, the amount of watery vapour is increased, and the chemical

composition is altered. Expired air contains 100 times more carbonic acid, a small amount of nitrogen, and nearly 5 per cent. less oxygen. The object of ventilation is to remove this expired air and replace it by pure supplies. To do this, however, under the usual thermometric condition which prevails in the country, without warming the incoming air, would cause the temperature of the byre to fall below that, *i.e.*, 60° Fahr., which is generally regarded by agriculturists as the most favourable for the forced production of milk. Consequently, no matter what system of ventilation is adopted, the attendants persist in closing all openings in order that the heat of the animal be retained, with the result—a more or less polluted atmosphere.

ANNUAL REPORT FOR 1900 FROM THE PRINCIPAL OF THE ROYAL VETERINARY COLLEGE TO THE ROYAL AGRICULTURAL SOCIETY.

Research Laboratory.

During the year 1900 343 morbid specimens were forwarded for examination to the laboratory, which was established at the Royal Veterinary College in 1890 for research in comparative pathology and bacteriology, and which has since been maintained by the aid of an annual grant of £500 from the Royal Agricultural Society. The number of specimens similarly forwarded in 1899 was 380, and in the preceding year 327. The diseased parts and organs sent for examination represented a great variety of morbid conditions, but a large proportion was furnished by the bacterial diseases. In many cases microscopic examination and cultural and inoculation experiments were required to determine the nature of the disease, and by such means the cause of many obscure cases has been cleared up.

In addition to the experiments forming part of the series instigated by the tuberculin sub-committee of the Society, a number of special experimental investigations intended to throw light on problems in pathology or therapeutics have been carried out. The results of some of these will be referred to in this report.

Anthrax.

The published returns of the Board of Agriculture show that during the year 1900 57 outbreaks of this disease were reported. Judged in this way, the disease was rather more prevalent than during the preceding year, in which the outbreaks numbered 53; but, on the other hand, the number of animals actually attacked was less in 1900 than in 1899, the respective figures being 952 and 950. It will be observed that in each of these two years the average number of animals attacked in an outbreak was less than two, and the fact may be taken as evidence of the beneficial effect of the regulations which are now enforced against this disease. Anthrax is not a contagious disease in the ordinary sense of the word, but in the absence of certain precautions it may within a very short space of time attack a large number of animals and appear to spread by contagion. Although the average number of animals attacked in outbreaks in this country is less than two, in occasional cases the number is very much higher, and with rare exceptions these more serious cases are the result of ignorance or carelessness in the management of the animals.

It is a well-known fact that the anthrax bacillus which is the cause of the disease is very resistant to heat and cold, and is also very resistant to disinfectants. It is also very resistant to the action of some of the most powerful disinfectants, and is therefore a very dangerous to its

fellows in the same building or field while it is still alive, because of the germs which are apt to be passed out with the excrement or urine; but it may become an infinitely greater source of danger after death if, without any precautions in the way of disinfection, the carcase is skinned or dressed. As a rule every drop of blood in an animal dead of anthrax swarms with anthrax bacilli; and to skin or dress an anthrax carcase means the liberation of countless numbers of these germs, with the possibility that they may infect the hands of those who conduct the operation, or give rise to further cases of anthrax in animals by being taken in with food or water.

These considerations show the necessity for isolating animals suspected of anthrax, and for great care in disposing of the carcases of animals dead of the disease. The sole duty of the agriculturist when he suspects the existence of anthrax among his animals is to give information to the police, and if the suspicion is verified the officers of the Local Authority will take over the duty of disposing of the carcase in the proper manner. On no account should an animal suspected of anthrax be bled, and the carcase of such animals should not be skinned or mutilated in any way.

The necessity for these precautions has often been insisted upon in previous reports, and the Society has issued a leaflet with the object of giving the fullest publicity to them. Nevertheless, many reports of gross neglect of such precautions continue to reach the Laboratory. One of the most frequent examples of such neglect is the feeding of pigs with the flesh of cattle or sheep that have been unexpectedly found dead. This is a double offence, first, because it generally involves the skinning of the carcase, with consequent risk to the person who does it, and serious contamination of the ground; and, secondly, because it is a very certain method of infecting the pigs so fed with anthrax. It cannot be too strongly impressed upon farmers that animals, particularly cattle, that are unexpectedly found dead, or which die after a very brief illness, for which no cause is apparent, ought to be suspected of anthrax; and, unless it is known that the cause of death was not anthrax, raw flesh ought not to be used for feeding pigs.

Inquiries which were made during the past year have proved that in many parts of the country the feeding of pigs with the raw and often putrid flesh of cattle, sheep, or horses, that have died from disease, is a comparatively common practice. Apart from the revolting aspect of such a diet for animals intended for human food, the habit deserves to be strongly condemned, owing to the fact that it lends itself to the dissemination of disease.

What has been said above makes it evident that, given one case of anthrax in a herd or flock, the disease may easily spread to a large number of animals, but it does not explain the origin of the first case in an outbreak.

As a matter of fact, this point is scarcely ever cleared up in the case of outbreaks occurring among animals at pasture, though it is highly probable that the starting point of most such outbreaks is water used or ground remaining contaminated from the blood or excreta of a previous case of the disease, which may have occurred years before. When, indeed, in cases involving the spreading of anthrax bacilli is spread on the surface of the ground, the germs may soon perish from the effect of sunlight and frost, or in some instances may be destroyed by their growth; but in certain favourable circumstances, such as high moisture the bacilli resist these influences, and remain viable for a long time. The most serious form of soil contamination is that in which the germs are protected by a plant, may resist all vicissitudes of weather, and remain viable for a long time until, when accidentally exposed, they give rise to a new case of the disease.

Although it is thus probable that a new case of anthrax may arise in a local, it is certain that a water supply, or a source of infection, is essential. This is probably always the case, and the only way of preventing a new case is to

occur among animals kept in towns, and fed exclusively on dry forage. In a former report, reference was made to an outbreak of this kind in a London stable. In that case it was proved experimentally that the infection was conveyed by means of the foreign oats on which the horses were being fed, and the owner of the horses thus infected obtained damages from the seller of the oats. During the past year several samples of cake and oats were sent to the Laboratory to be tested, in consequence of a suspicion that the material in question had caused anthrax among animals fed therewith. Although in all these cases it was reported that the circumstantial evidence pointed very strongly to the presence of anthrax germs in the cake or oats, the experimental tests gave negative results in all but one case. In that instance it was ascertained that anthrax germs were undoubtedly present in the sample of oil-cake sent for examination.

It is not improbable that this is a more frequent cause of anthrax in this country than has hitherto been supposed, and, at any rate, it is a possible cause that ought not to be lost sight of when an outbreak of the disease occurs among stall-fed animals of which cake forms part of the diet, and, especially, in those cases in which there is no history of a previous attack of anthrax on the same premises or farm.

Glanders.

An unsatisfactory feature of the returns of the Board of Agriculture regarding the occurrence of the contagious diseases of animals during the past year is the very considerable increase in the prevalence of glanders which they disclose. The number of outbreaks reported during the twelve months was 1126, and the number of horses attacked 1865, while the figures for the preceding year were 853 and 1472 respectively.

It was hoped that with the discovery of mallein, the immense value of which as a diagnostic agent is now universally admitted, marked progress towards the extermination of the disease would soon be made. This expectation has not been realised, and the explanation is not difficult to find.

At present the law does not regard a horse as glandered unless outward or clinical signs of the disease are present, and in the administration of the Glanders Order a quite unusual interpretation is given of the word "suspected." For example, in the case of pleuro-pneumonia or swine fever the word "suspected" covers all animals that are known to have been recently exposed to the contagion of these diseases, but in dealing with outbreaks of glanders the practice of most local authorities is to consider "suspected" only such horses as present doubtful external symptoms of glanders. As soon as these and the obviously glandered animals have been destroyed and the premises have been disinfected, the outbreak is considered to be at an end, and no restrictions are placed on the movements of outwardly healthy-looking horses, although it is known that they have been exposed to the contagion of glanders. The result is that such horses are freely sold, and that the disease is thereby carried into new stables. It is even possible that at the present time the confidence which is placed in mallein is contributing to the spread of the disease, unscrupulous owners applying the test to their apparently healthy horses, and promptly disposing of those that react. To counteract this state of affairs some amendment of the law is urgently required.

It is true that in the present distribution of the disease glanders is not of great practical interest to the agriculturalist, since in the main it is a disease of town horses. At the same time, however, the extension of the disease cannot be viewed with indifference by farmers, for as long as it is a common disease in the large cities there is considerable risk of the infection being carried into country places. A number of the horses annually "cast" from the large railway, omnibus, and tramway studs find their way back into the

country to work on the land, and it cannot be too well known that whoever buys such a horse takes considerable risk of importing glanders. Where the circumstances justify any suspicion of that kind, the newly purchased animal ought to be tested with mallein before he is put in association with other horses.

Foot-and-Mouth Disease.

After five complete years (1895-99) of freedom from foot-and-mouth disease, this dreaded plague reappeared in the British Islands in the early part of the past year, and although, fortunately, it has not yet attained to serious dimensions, the contagion has been carried over a large part of England, and has defied the efforts of the Board of Agriculture to stamp it out. The present invasion of the disease is unlike any of the previous visitations in the matter of the number of outbreaks in relation to the time over which they have been spread. The long period from 1839 to 1886 covered the duration of what may be called the first visitation, though there is some reason to believe that the country was free from the disease for a few months at the close of 1879, and that the outbreaks of 1880 were started by importation of diseased animals from abroad. The number of animals affected during this first visitation was probably not far short of seven millions, and in one year alone (1871) the officially reported outbreaks were over 5000 and the number of animals attacked over half a million. In 1886 only one outbreak of the disease was detected, this representing the last spark of the conflagration which was lit in 1839. From 1887 to 1891 inclusive the country was entirely free from the disease, and the second visitation began in February 1892, when the disease was detected among foreign cattle exposed in the Metropolitan Cattle Market. In spite of all the efforts made to check the spread of the infection, the disease soon appeared in many places widely apart, but it was apparently eradicated by the month of June, after nearly 5000 animals, in ninety-five separate outbreaks, had been attacked.

The third invasion comprised two outbreaks in 1893, one of which was detected in a London cowshed on 1st January, and the other near Hastings on 17th February. As in the case of the subsequent invasions, the source of the outbreaks of 1893 was not traced, but it had to be set down to mediate contagion from abroad, since the importation of foreign animals, save for slaughter at the port of landing, came to an end in 1892.

In 1894 the disease reappeared in the same mysterious manner, but this, the fourth, visitation comprised only three outbreaks, and involved only 268 animals.

After five years of freedom from the disease the fifth invasion was started with an outbreak which was detected in Suffolk during the latter part of January 1900. The second outbreak occurred in Norfolk in February, and during the same month the disease was detected in Bedfordshire. A month later it reappeared in Norfolk, and in May it was detected in Hertfordshire. No fresh outbreaks were reported in June or July, and it was hoped that the disease had been stamped out, but in August it cropped up again in places as far apart as the East Riding of Yorkshire and Flintshire in North Wales. In September it was detected in Wiltshire, and in October in Staffordshire. Again there was an interval of two months without any fresh outbreaks, but in December an outbreak was discovered in Essex, and at the date of writing the disease has been carried over into the new year by an outbreak in Suffolk.

The returns published during the last week of 1900 show that the total number of outbreaks detected during the year was twenty-one, with 266 animals attacked. As already said, no previous visitation has had such long intervals between the separate outbreaks, and it is natural to search for some explanation of the erratic course of the disease during the past year.

It is impossible to doubt that this invasion, like those of 1893 and 1894, was started by germs of the disease imported from abroad through the agency of some intermediary bearer. The facility with which the disease may be spread in this way is well recognised in veterinary literature, and attested by many striking examples; and the opportunities for the carriage of infective materials provided by the daily traffic with parts of the Continent in which foot-and-mouth disease has for some time been rampant, make the view that it was imported into this country in January last perfectly plausible. Nor is it surprising that the investigations carried out by the Board of Agriculture failed to trace the primary source of the contagion; but the same can hardly be said of the mystery which hangs over the twenty separate outbreaks which occurred between February and December.

It is not credible that each of these was due to separate importations of the virus from abroad, and although no connection between the successive outbreaks has been traced it is almost certain that such a connection existed. The probability is that the recorded outbreaks of last year form part of a continuous chain of which a good many of the links have never come under the notice of the Board of Agriculture. That a considerable number of outbreaks have not been reported or officially discovered may be set down in part to ignorance of the symptoms of the disease on the part of stockowners, but it is to be feared that wilful concealment of the disease is part of the explanation. More vigilant inspection, and the infliction of the fullest penalties allowed by the law on convicted offenders, are the only remedies for wilful concealment, and an endeavour to counteract the other source of difficulty in following up the disease must be made by diffusing among farmers and others a better knowledge of the symptoms of foot-and-mouth disease. Fortunately these are very characteristic.

The first symptoms generally appear within a week after the exposure to infection. In *initial* these are slight dulness, shivering, loss of appetite, "staring of the coat," and stiffness in movement. Even at this time the animal is in a state of fever, the temperature being generally two or three degrees above the normal. Within a few hours these premonitory symptoms of illness are followed by others which are characteristic of the disease. The animal ceases to feed, and is reluctant to move. From time to time it makes a peculiar snacking noise with its lips, from which more or less frothy slobber escapes. If the mouth be opened white blister-like elevations will now be found on some part of the lining membrane of the mouth, and these soon burst and leave shallow raw sores or ulcers. When made to move the animal is obviously lame or "tender on its feet," and while standing it frequently shifts the weight from one leg to the other, and shakes its feet, as if to get rid of something adhering to them. This tenderness of the feet is well marked even before the formation of blisters on them; but these soon appear on the skin between the claws or behind them, immediately above the hoofs.

When the vesicles have burst the animal suffers increased pain in the mouth, and it refuses to chew the cud, and owing to its inability to swallow without great pain the saliva is allowed to trickle from the mouth. The general appearance of the animal at this stage is indicative of great misery. The eyes are dull, the ears drooping, the back arched, and the hair matted and matted. The bowels are generally more or less constipated, and in such cases the secretion of milk is greatly diminished or almost arrested.

As a rule the animal remains in this condition for about a week, at the end of which it may recover, or it may die. At the end of that time the animal is usually found dead, and the sores in the mouth have not yet healed, the pain is so great that the animal is unable to attempt to eat,

especially if grass or other soft food is to be had. The tenderness of the feet gradually declines also, and in most cases all the visible symptoms of the disease have disappeared within ten or fourteen days after the onset.

The symptoms in *sheep* are generally somewhat different from those just described, inasmuch as in them the mouth often escapes attack, or is only slightly affected. On the other hand, in sheep the disease always attacks the feet, and the lameness is severe. The pain in the feet is so great that the animal is unable to gather its food, and it lies persistently, with consequent rapid loss of condition.

Pigs are affected in much the same way as sheep: there is always pronounced lameness or tenderness of the feet; and although blisters may form on the snout or in the mouth, they are not so constant in that position as in cattle.

As has already been said, these symptoms are very characteristic, but it would not be correct to say that the diagnosis of foot-and-mouth disease never presents any difficulty. It may, however, be said with truth that there is never any difficulty in recognising the disease when any considerable number of animals are simultaneously affected, at least in the case of cattle. The markedly contagious character of the disease is a point of great importance in diagnosis, as is also the fact of its affecting simultaneously both cattle and sheep. Conversely, no matter how close the symptoms presented by a single animal may resemble those described above, the disease may be set down as not foot-and-mouth disease when, in spite of abundant opportunities for infection, it does not spread after a few days to other cattle or sheep. Doubts are only justified when, as in the case of a single diseased animal seized in a market, there is no evidence that the condition is contagious. Even then, of course, it is wise to provisionally diagnose the affection as foot and mouth disease, and not to wait for evidence of the disease being contagious before taking the steps which the law directs. It is hardly necessary to observe that the duty of determining whether a given animal is the subject of foot and mouth disease or not is not laid upon the stockowner. Whenever there are any reasonable grounds for suspicion, and the owner is not certain that the symptoms exhibited by any of his animals are not those of foot and mouth disease, his duty under the law is to give immediate notice to a constable of the local police force.

Although it is in the highest degree desirable that such intimation should be given with all practicable speed by the owner of any animal which presents such symptoms as those previously described, leaving the responsibility of making an exact diagnosis to the veterinary inspection of the local authority, it is not necessary or desirable that the owner should report every case of disease affecting the mouths or feet of his animals as a suspected foot and mouth disease. In cattle the combination of symptoms of the mouth and lameness ought always to be reported for there is no other disease in which that combination is present.

Diseases of Pigs caused by Foot and Mouth Disease

As already stated, foot-and-mouth disease is a very infectious disease, and there are three other diseases of the same kind which are present in connection with the foot-and-mouth disease. That disease is *underrunning*, *scald*, and *scabby*. These diseases are like the rapidity of foot-and-mouth disease, and are generally contracted by animals pastured with the affected animals. *Underrunning* is a disease which is primarily a disease of the feet of the animal, and is characterised by a swelling on the part of the foot which is affected, and by a pain which is very severe in foot-and-mouth disease. The disease is generally contracted by animals which are generally towards the feet, and is characterised by a swelling on the part of the foot and the sensitive structure of the foot.

the sheep is generally lame or tender in the whole of its feet, while in foot-rot the disease may for a long time be confined to one foot.

The second disease of the sheep which has some resemblance to foot-and-mouth disease in respect of the situation of the lesions is the one which is known as malignant aphtha. The resemblance is certainly not at all close, and even laymen need experience no difficulty in distinguishing between them. Malignant aphtha does not attack the feet, and it is usually met with as a troublesome affection of ewes and young lambs. In the case of the lambs the disease is manifested by the formation of sores on the nose or lips, and the ewes develop similar sores on the teats and udder. In neither case are the sores ever blister-like: they are generally covered by brownish crusts or scabs, and in the lambs they do not form in the interior of the mouth, but on the outside of the lips or around the nostrils. The disease apparently is only spread by direct application of matter from the sores to the skin, and it is rarely seen affecting lambs more than a few weeks old.

The third disease of sheep which has been mistaken for foot-and-mouth disease is one that has not received much attention in veterinary literature, although it is not of very rare occurrence. It is the one which led to a mistaken declaration of the existence of foot-and-mouth disease in Perthshire in the month of September last. In Scotland it has long been familiar to many shepherds, and it has various local designations, the commonest of which is perhaps "orf." It attacks lambs of any age up to a year old, and it is rarely seen in adult sheep. The sores which characterise it are not unlike those of aphtha, and the parts on which they usually form are the face and legs. On the face the lesions are present on the skin around the nostrils, and on the hairy surface of the lips—never in the interior of the mouth. On the legs the sores may form anywhere between the top of the hoof and the knee, or even higher, and sometimes the sensitive structures around the top of the claws are also involved, with more or less lameness as a consequence. The disease has never been known to spread to cattle or other animals than sheep.

It has been thought well to give this outline account of the diseases which may be, and some of which actually have been, mistaken for foot-and-mouth disease, but the best advice to the layman with regard to the differential diagnosis of affections of the mouths and feet of cattle or sheep is to free himself from all responsibility by reporting to the authorities every such condition about which he feels any doubt. It is much better that the Board of Agriculture should receive twenty false alarms than that a single genuine outbreak of foot-and-mouth disease should remain unreported for even a day after its discovery.

Swine-Fever.

During the past year the total number of outbreaks of this disease dealt with by the Board of Agriculture was 1940. This represents a decrease of 382 outbreaks as compared with the previous year, but it is only 215 less than in 1897. The present state of affairs is no doubt an improvement on that which existed before the disease was taken in hand by the central authority, but the comparatively slight impression made on the disease during the last three years is somewhat disappointing, since it does not appear to justify much hope that it will ever be exterminated by the present methods of dealing with it. A notable feature of the returns is the smaller average number of pigs slaughtered in each outbreak as diseased or exposed to infection as compared with previous years. The average number thus destroyed in each outbreak during the past year was nine, while in 1897 it was eighteen. This can hardly be explained otherwise than by supposing that the circumstances which constitute "exposure to infection" are now judged more leniently than they were a few years ago.

Rabies.

It appears probable that the past year will remain memorable as the one in which rabies was eradicated from Great Britain. As a matter of fact, no case of the disease was detected in England or Scotland during the past twelve months, and the only cases reported during the year were in Wales. The total number of dogs attacked was six, but five other animals were destroyed in consequence of having been bitten by rabid dogs. No case of the disease has been detected in dogs since the second week of October last.

DISEASES CAUSED BY ANIMAL PARASITES.

Parasitic Gastritis in Cattle and Sheep.—Former reports have contained references to the very serious losses caused among both cattle and sheep by minute, almost microscopic, worms which inhabit the fourth or true digestive stomach of these animals. Judged by the number of specimens forwarded to the research laboratory, this form of disease has been less prevalent among sheep and lambs in 1900 than in some recent years. It is not improbable, however, that the smaller number of such cases that came under notice during the past year is mainly due to the fact that, as an outcome of the investigations which were carried out a few years ago, the disease is now much better known both to farmers and to practising veterinary surgeons, with the result that a correct diagnosis is more frequently made without having recourse to the laboratory. During the year a number of new examples of the mischief which these stomach-worms cause in cattle came under notice, and advantage of the opportunities thus presented was taken to carry out some further experiments with regard to treatment.

During the year three cows from three different farms on which this disease was found to exist were brought to the Royal Veterinary College. All of these at the time of their admission were at a rather advanced stage of the disease, and the drug which was selected for experimental trial on them was thymol, as within the last few years it has acquired considerable reputation as a remedy against round worms located in the intestines. Unfortunately, it has to be confessed that it entirely failed to produce any favourable effect on the course of the disease in any of the three animals, the condition of which steadily advanced from bad to worse. One of them had to be destroyed owing to weakness and emaciation, and the other two, though still alive at the time of writing, are much worse in appearance than when they were first put under treatment. The maximum quantity of thymol administered to one animal was $2\frac{1}{2}$ ounces, this being given in five equal doses spread over a period of twenty-five days. It is doubtful whether much larger doses could be given with safety.

As has been pointed out on former occasions, the great difficulty of effecting a cure in this disease lies in the fact that it is scarcely possible to insure that any medicine administered by the mouth shall reach the fourth stomach in such a degree of concentration as will prove fatal to the worms there. The same remark applies to the treatment of parasitic enteritis in the horse. One may administer a medicinal substance in quantities approaching the fatal dose, and in such a degree of concentration as to insure the immediate death of any worm immersed in it, but by the time the drug reaches the fourth stomach in the case of ruminants, or the large intestine of the horse, it may have become diluted or absorbed to such an extent as to render it quite innocuous to the parasites which it is intended to kill. In this connection it may be mentioned that the favourable results which have been claimed for thymol in the treatment of parasitic inflammation of the intestine in colts have not been borne out by some experiments which were made with it during the

spring of last year, although it was given in much larger doses (half ounce) than those generally recommended.

There is reason to think that parasitic gastritis in cattle is still pretty frequently mistaken for tuberculosis during life, though such a confusion is not possible if the body is opened after death. Experience justifies the statement that in cattle the most frequent cause of persistent diarrhoea, with steady loss of condition in spite of unimpaired appetite, and without cough or other evidence of lung disease, is the presence of worms in the stomach. Diarrhoea is far from being a constant symptom in tuberculosis, even at advanced stages of the disease; and before it sets in there is generally distinct evidence of lung disease, and the appetite is much impaired. After death a mistake is not possible, for the *post-mortem* examination of an animal which has died from tuberculosis, or which has been obviously ill from that disease before it was killed, always reveals very gross signs of disease in the internal organs, usually in both chest and abdomen. On the other hand, at the *post-mortem* examination of animals that have died from parasitic inflammation of the stomach, the whole of the internal organs, with the exception of the fourth stomach, are generally quite normal in appearance, and even the stomach might pass for healthy to casual inspection.

In the absence of any known means of curing this disease, preventive measures acquire great importance. The chief of these are isolation of suspected animals indoors, to prevent further contamination of the soil with the eggs and young worms, and destruction of their manure.

Æstrus disease in sheep.—There does not appear to be any popular name for this disease, probably because it is, fortunately, not of very common occurrence in this country. It is caused by the larvæ of a species of gadfly—the *Æstrus ovis*—which inhabit the so-called air-sinuses of the head and also the horn-cores in horned breeds. The flies attain maturity about midsummer, and in the months of July and August the females deposit their eggs, or, as some suppose, the already hatched larvæ, about the nostrils of the sheep. The minute larvæ make their way up the nasal cavities, from which they pass to the air-sinuses and the horn-cores, to attach themselves to the membrane lining these cavities. There they remain till the following spring, and they then migrate back into the nasal cavities on their way to the exterior, their outward passage being assisted by the sneezing efforts provoked by the irritation of the mucous membrane. The larvæ on reaching the soil speedily pass into the rigid, motionless "nymph" stage, from which, after six or eight weeks, the fully formed fly escapes.

As a rule the larvæ do not give rise to any symptoms until they begin their migration from the air-sinuses back into the nose, which in this country usually occurs about the month of May, and even then they do not occasion any noticeable disturbance unless they are numerous.

During the past year very serious losses among sheep were caused by this parasite in Wales, and a number of examples of its effects were forwarded to the Laboratory by Mr W. Williams, M.R.C.V.S., Llandilo. The principal symptoms exhibited were as follows: Blood-shot staring eyes, elevation of the head, staggering gait, a discharge of matter from the nose, sneezing, and coughing. Great numbers of sheep died, and many others were killed, owing to their apparently hopeless condition. In those cases in which death has been produced, the fatal effects have generally been attributed to the intense irritation of the lining membrane of the nose and sinuses which the larvæ occasion. But it was observed that in the specimens sent from Wales these nasal lesions were accompanied by more or less extensive pneumonia, and it is not improbable that the latter condition was the main factor in determining death. In one case a considerable number of the larvæ had migrated into the wind-pipe; but, apart from such a cause of lung complications, the entrance into

the air tubes of purulent matter from the nasal cavities would probably be sufficient to account for the pneumonia.

Recommendations with regard to the treatment and prevention of this disease are not lacking in veterinary text-books, but it must be confessed that some of them are counsels of perfection, and the others are of very doubtful value. To the former class belongs the advice to keep the sheep away from the neighbourhood of woods and plantations which are specially frequented by the gadfly, and to destroy the larvæ after they have been expelled from the noses of affected sheep. Smearing of tar and other substances supposed to be distasteful to the flies around the nostrils of the sheep is not altogether impracticable, but there is no reliable evidence that the insects can be deterred from depositing their eggs or larvæ by such means. Dishorning or opening of the sinuses of the head by surgical operation has also been suggested in order to remove the larvæ, or to destroy them by introducing substances fatal to them; but, in the present state of knowledge, probably the best plan is to make mutton of all the affected animals which are in fair condition, as soon as the disease is recognised. As already mentioned, the disease is not very common in this country, but it is said that in the district in Wales in which so many deaths occurred last May a similar outbreak took place about twenty years ago.

Fowl Cholera.

Fowl cholera has long been known on the Continent of Europe as a dreaded scourge of the poultry yard. Historically, it is of great interest, because it was one of the first diseases proved to be caused by bacteria, and because the late M. Pasteur's researches in connection with it conducted him to a discovery which may be said to have formed the foundation of the method of protective inoculation by means of artificial cultures. The organism which is the cause of the disease is called the bacterium or bacillus of fowl cholera, and it is so minute that it appears as a mere point, just visible to the eye, when it is magnified 500 times. To appreciate its exact form, at least double that magnification is necessary. In a bird dead from fowl cholera, the blood everywhere swarms with these bacteria, for it is mainly in that fluid that the germ multiplies when it gains access to the body. The bacteria, however, are not confined to the blood, for they are always present in great numbers in the intestines of diseased fowls. As the name indicates, profuse diarrhoea is one of the most constant and prominent symptoms of the disease, and countless numbers of bacteria are voided with the thin excrement passed by the affected birds. It is mainly, or entirely, by means of such excrement that the disease is spread, and, in view of the circumstances in which fowls are usually kept, and their habits of feeding, it is not surprising that the disease often causes terrible ravages when once it has obtained a footing in a poultry-yard.

English veterinary literature does not contain any authenticated case of the occurrence of fowl cholera in this country, and, probably on this ground, an eminent bacteriologist has even asserted that the disease does not occur in Great Britain. That, however, is an error, and since the foundation of the Research Laboratory at the Royal Veterinary College a considerable number of genuine outbreaks of fowl cholera have been investigated there. During the past year two such outbreaks came under notice. The two outbreaks occurred almost simultaneously on two farms several miles apart, and it was not apparent that there had been any opportunity for the carriage of infection from one farm to the other. The only discoverable circumstance that could be held to have had any possible bearing on the origin of the outbreaks was that at both farms the poultry were being fed with corn sweepings from foreign ships that discharged at a neighbouring port.

In these two cases, as in all the others that have come under notice at the

Laboratory, it was suspected that the fowls were being poisoned, but microscopic examination of the blood, which is a routine practice in such cases, left no room for doubt that the cause of death was fowl cholera. In each case the blood was found to be swarming with minute organisms having the size and shape of the bacteria of that disease, and artificial media inoculated with a trace of blood yielded cultures with the characters that belong to the germ of fowl cholera. Furthermore, experiments showed that these artificial cultures were capable of killing fowls within forty-eight hours after inoculation, the blood of such fowls swarming with the bacteria, just as in natural cases of the disease.

The discovery by M. Pasteur, to which allusion has been made above, was that artificial cultures of the fowl cholera bacterium may be successfully employed to vaccinate fowls against the natural disease. Inoculation with cultures that have only been grown for a few days has just the same effect as inoculation with a drop of blood from a bird dead of fowl cholera—that is to say, it sets up a fatal attack of the disease. On the other hand, if the incubation of the artificial culture is continued, with free exposure to the air, the bacteria gradually lose their virulence, and become “attenuated,” and such attenuated cultures may be employed to confer on fowls a valuable degree of immunity against the disease. Although the feasibility of this method is well established, it has never been put into practice on a large scale. In the first place, the expense of the vaccin and of the operation is a barrier to the method in dealing with animals of such small value as fowls; and in the second place, before the disease has been correctly diagnosed it has often made such headway that the majority of the fowls on the premises are either dead or infected.

Although the origin of the first case of fowl cholera cannot always be determined, there are outbreaks in which the mischief can be clearly traced to some newly purchased fowl, which suggests that, just as in the case of the larger animals of the farm, it is well to place newly bought poultry under quarantine for a few days.

The chief symptoms of fowl cholera are great dulness, loss of appetite, separation from the rest of the poultry, livid discoloration of the comb, drooping of the wings, ruffling of the feathers, and diarrhoea. When such symptoms are exhibited by a number of fowls, or when a number of fowls die suddenly, even if these symptoms have not been noticed, it is well to suspect fowl cholera. As soon as the disease has been diagnosed, every ailing fowl ought to be destroyed, and the survivors ought, if possible, to be removed to fresh premises while the old ones are thoroughly cleansed and disinfected. If the weather permits, the best plan is to keep the fowls in the open, and to feed them on a fresh plot of ground every day. In short, the measures to be taken are those that are dictated by a knowledge of the fact that the disease is spread by food or water soiled with the excrement of the affected birds.

BRITISH CONGRESS ON TUBERCULOSIS.

This Congress will be held in London, from Monday, 22nd July, to Friday, 26th July.

Every British Colony and Dependency is invited to participate by sending delegates; while the governments of countries in Europe, Asia, and America are invited to send representative men of science, and others, who will be the distinguished guests of the Congress.

The object of the Congress is to exchange the information and experience gained throughout the world as to methods available for stamping out the

disease. Papers will be read, and clinical and pathological demonstrations will be given; while the museum, which is to be a special feature of the Congress, will contain pathological and bacteriological collections, charts, models, and other exhibits.

Authorities in this and other countries will be invited to supply documents bearing upon the historical, geographical, and statistical aspects of the subject; while as a result of the papers and discussions, practical resolutions will be formulated which will serve to indicate the public and private measures best adapted for the suppression of tuberculosis.

The work will be divided into four sections as follows:

Section I.—State and Municipal.

Section II.—Medical, including Climatology and Sanatoria.

Section III.—Pathological, including Bacteriology.

Section IV.—Veterinary (Tuberculosis in Animals).

The officers of the last of these sections are: *President.*—Sir George Brown, C.B., Bryn Hyfryd, Harrow. *Secretaries.*—Prof. Hobday, F.R.C.V.S., 27 Lower Phillimore Place, Kensington, W.; Prof. Bradley, M.R.C.V.S., Royal (Dick's) Veterinary College, Edinburgh; Prof. H. Woodruff, M.R.C.V.S., Royal Veterinary College, Camden Town, N.W.

The Congress will consist of Honorary Members, Delegates, and Members. Honorary Members will be persons nominated by any foreign Government or University, or by the Executive Committee, and shall not pay any subscription whatsoever. Delegated and Ordinary Members will be other persons, British, Colonial, or Foreign, who may wish to attend, including representatives from Governments or institutions within the British Empire. Delegated and Ordinary Members will receive their cards on forwarding the sum of £1 to the Secretary-General of the Congress, 20 Hanover Square, W. The holder of a ticket of membership is entitled to admission to all the meetings of the Congress, and to receive the "Transactions" and other publications. Tickets to social gatherings and excursions will be allotted in order of application, or by ballot if necessary.

As the expenses of the Congress will be very considerable, Donations to the Reception Fund are earnestly invited. Donations of more than one guinea will be considered as including Member's subscription, and will entitle the donor to all privileges of Membership. Donations from Corporate Bodies may include the subscriptions of representatives.

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**A FURTHER CONTRIBUTION TO THE PATHOLOGY
OF AFRICAN HORSE-SICKNESS.**

By J. M'FADYEAN, Royal Veterinary College, London.

In an article which appeared in the preceding volume of this journal (Vol. XIII. p. 1), I published an account of a series of experiments which had been carried out at the Royal Veterinary College with the object of throwing further light on the nature of this disease. The results of these experiments corroborated a number of important points with regard to the pathology of the disease which had been previously established by the researches of Dr Edington, but they also established some new points in connection with the nature of the infective agent, the possible methods of infection, and the lesions commonly present in animals dead of horse-sickness, viz. :—

1. That the so-called *œdemamyces*, which Edington had found in horse-sickness blood, could not be regarded as the cause of the disease.

2. That horse-sickness blood is not deprived of its infective property by filtration through a Berkefeld or a Chamberland filter.

3. That the most constant of the lesions present in animals dead of horse-sickness is an intense congestion of the mucous membrane of the right half of the stomach.

4. That the administration of a large dose of horse-sickness blood by the mouth is a very certain method of communicating the disease.

During the past twelve months some further experiments have been carried out, and these will be here described.

THE EFFECT OF DESICCATION ON THE VIRUS OF HORSE-SICKNESS.

Some experiments bearing on this subject have already been published by Edington.¹ In one of these citrated blood (about sixteen of blood to one of a 10 per cent. solution of neutral citrate of potash), was first dried, and when thoroughly dry it was reduced to powder and placed in a drying chamber for forty-eight hours. The temperature at which this desiccation was effected is not stated in the account of the experiment. Two grammes of the dried blood was dissolved in sterile salt solution and injected under the skin of a horse. A second experiment with blood from another case of horse-sickness was carried out at the same time. In each case the only result was a slight rise of temperature about two hours after the inoculation.

In another experiment two horses were inoculated with two grammes each of blood which had been dried first under the shade of a verandah, and after that in a drying chamber. The sojourn in the drying chamber was for twenty-four hours, but the temperature is not stated. In each of the two horses febrile disturbance followed the inoculation with the dried blood, and, although a later inoculation with virulent blood showed that the animals had not been fully protected, Edington concluded that this disturbance was due to the virus of horse-sickness.

These experiments may, therefore, be summed up by saying that the first two appeared to indicate that the agent of infection of horse-sickness is deprived of its vitality by an exposure of forty-eight hours in a drying chamber, and the last that it still retains its vitality after similar desiccation for twenty-four hours. In the following experiment the result was quite unambiguous.

Experiment I.—On the 7th November last 2 cc. of blood from a horse dead of horse-sickness on that date was spread on the bottom of a sterile Petri dish, and placed in an incubator at 37° C. The Petri dish was four inches in diameter, and the blood therefore formed only a thin layer on the bottom of it. At the end of twenty-four hours the blood had assumed the appearance of a thin, dry, adherent film. On the 13th November the dish was taken out of the incubator, and from that date until the 20th it stood at the temperature of the laboratory. On the latter date the thin film of blood was scraped off the glass and triturated in a mortar with 10 cc. of bouillon. The whole of the liquid was then injected under the skin of the neck of a pony. The morning and evening temperature of the animal from the second day after the inoculation are shown below.

			A.M.		P.M.
Nov.	22	.	100·8	.	100·6
"	23	.	100·2	.	100·2
"	24	.	100·4	.	99·6
"	25	.	99·2	.	99·4
"	26	.	99·4	.	101·2
"	27	.	102·6	.	102·4
"	28	.	103	.	103
"	29	.	104	.	104·8
"	30	.	103·6	.	105
Dec.	1	.	99·2	.	(Killed)

¹ Vol. XIII. of this journal, p. 213.

On the morning of the 1st December the temperature was found to be sub-normal, and death appeared to be imminent. The expression was anxious, the conjunctival membrane was congested and œdematous, the respirations were hurried, and the animal staggered when made to move. At 11 A.M. it was chloroformed to death.

Autopsy.—No trace of reaction at the seat of inoculation. Peritoneal cavity contains about half-a-pint of straw-coloured watery liquid. The stomach contains a considerable quantity of food materials. Its right half is most intensely congested. Regarded from a distance it is of a port-wine tint, but when more closely inspected the membrane is seen to be closely beset with small hæmorrhages. There is some congestion of the duodenum, but the rest of the intestinal tube is normal. Abdominal lymphatic glands normal. Spleen weighs $2\frac{1}{2}$ lbs.; pulp normal in appearance. Liver and kidneys very full of blood. A few ounces (estimated at 6) of straw-coloured liquid in the pleural sacs. Lungs slightly œdematous. Pericardium contains 550 cc. (measured) of limpid straw-coloured watery liquid. Heart normal, save for some endocardial hæmorrhages on the wall of the left ventricle. Tongue and throat normal. No bacteria to be seen in cover-glass preparations of blood stained with methylene blue or fuchsin.

This experiment proves conclusively that the agent of infection of horse-sickness is not destroyed by very thorough desiccation at the body temperature.

THE EFFECT OF HEAT ON THE VIRUS.

Experiment II.—On the 21st July last 10 cc. of citrated blood plasma, which had been taken with aseptic precautions from a horse dead of horse-sickness on the 28th of February, was placed in a sterile test tube, which was then hermetically sealed and heated in a water-bath for ten minutes at 55°C . On the same date $2\frac{1}{2}$ cc. of the heated plasma was injected under the skin in front of the near shoulder of a pony. The temperature of the animal on the following days is shown below:—

July 21 . . .	101.4	July 26 . . .	101.8
" 22 . . .	101.4	" 27 . . .	102.5
" 23 . . .	101.2	" 28 . . .	103.5
" 24 . . .	100.8	" 29 . . .	104.2
" 25 . . .	101	" 30 . . .	104

On the afternoon of the 30th July the temperature fell to 101°F ., and the pony died at 2 A.M. on the following day.

Autopsy.—No liquid in pleural sacs and lungs not œdematous. Pericardium contains about half-a-pint of clear straw-coloured liquid. Right half of stomach intensely congested. No lesion at seat of inoculation.

Experiment III.—The material used for this experiment was exactly the same as that employed in the preceding one, save that the plasma was heated for ten minutes at 75°C . During the heating the plasma became converted into a solid coagulum, and to prepare the latter for injection it was triturated in a sterile mortar with an equal volume of sterile water. A pony was inoculated in front of the near shoulder with 5 cc. of the mixture.

The animal's temperature was taken daily after inoculation till the 30th July, on which date the companion pony inoculated with the same quantity of plasma heated to 55° C. died. As during these nine days the highest temperature registered was 101·8 it was concluded that the animal had not become infected, and the taking of the temperature was therefore discontinued. It was found dead in its box on the 15th of August, that is to say, twenty-five days after inoculation. To the groom who fed and watered it it had appeared to be quite well on the morning of the 15th August.

Autopsy.—Peritoneum and exterior of abdominal viscera normal. No liquid in peritoneum. Mesenteric glands normal. Spleen and kidneys ditto. Liver appears to be slightly fatty. The mucous membrane of the villous half of the stomach is intensely inflamed, and of a deep red colour. Mucous membrane of small intestines normal, except for a few small spots of congestion. Large intestine normal. Bladder distended with urine, but normal. Pleural sacs contain clear, yellow fluid (about a quart); pleura itself normal. Lungs a little œdematous, with a great quantity of frothy material in the trachea and bronchi, and pink in colour. Two or three ounces of yellow fluid in pericardium. Heart normal.

The conditions disclosed at the *post-mortem* examination left scarcely any doubt that the subject of this experiment had died from horse-sickness, but, nevertheless, it was thought well to place that beyond dispute. The conclusive evidence on this point is furnished by the result of Experiment XII.

This experiment proves that the virus of horse-sickness is not entirely destroyed by an exposure of ten minutes to a temperature of 75° C. The prolonged period of incubation is somewhat remarkable, but Edington has recorded an experiment in which twenty-three days elapsed between inoculation and death when the infective material was blood containing sulphuric acid in the proportion of 1 in 800. The delay in the evolution of the disease in these cases may have been due either to a process of attenuation of the hypothetical bacteria or to actual destruction of a large number of them. In a control experiment with unheated plasma the temperature began to rise on the seventh day after inoculation, and the animal died on the ninth day.

THE EFFECT OF PUTREFACTION ON THE VIRUS.

Experiment IV.—A flask of horse-sickness blood which had been taken direct from the carotid artery on the 28th February 1900, and which had since stood at the temperature of the laboratory, had a quantity of garden earth added to it on the 16th March 1900. It was then placed in an incubator at 37° C., and left there till the 28th of the same month (owing to an oversight it was left out of the incubator for the greater part of a day during this period). On the 28th March the intensely putrid mixture of blood and earth was mixed with an equal volume of tap water, and filtered through a Berkefeld filter. On the 29th March 20 cc. of the filtrate was injected under the skin in front of the near shoulder of a pony. The temperature of the animal on the 28th March was 100·6°, but through

an oversight it was not recorded for the next two days. During subsequent days it was as follows:—

March	31	.	.	.	.	.	102·5
April	2	.	.	.	.	.	105
"	3	.	.	.	.	.	105
"	4	.	.	.	.	.	105·4
"	5	.	.	.	.	.	98·4

The pony died on the 5th April at 6.30 P.M. During the greater part of that day it had been lying down and looking uneasy, but it was eating hay at 5 P.M.

Autopsy.—There is no reaction under the skin at the point of inoculation in front of the near shoulder, but between the muscles on both sides of the neck, and most marked on the right, there is a considerable amount of œdema of a gelatinous character. Some similar material is present along the course of the trachea. There is no measurable liquid in the peritoneal cavity, and the membrane itself is normal. Abdominal lymphatics normal. The stomach contains a moderate quantity of food materials; the greater part of the glandular mucous membrane is of a deep livid colour from congestion. Throughout the small bowel there are numerous areas of congestion, and there is also some congestion of the mucous membrane of the cæcum and colon. The spleen is of about normal size, and its pulp is fairly firm. Kidneys normal. Liver dark and full of blood. Bladder contains three pints of dark-yellow thick urine, which is found to contain a slight amount of albumen. The pleural sacs contain less than half a pint, and the pericardial sac about a pint, of clear, straw-coloured watery liquid. There is some gelatinous œdema around the roots of the lungs and the base of the heart. Heart and lungs normal. The throat and its lymphatic glands are normal, but there are some excoriations on the dorsum of the tongue. No bacteria to be seen in blood or gelatinous exudate in methylene-blue preparations.

Experiment V.—On the 6th April 1900 about a pint of blood from the subject of the preceding experiment was mixed with about eight times that quantity of garden earth in an earthenware pot. The pot was placed in a conservatory of which the temperature fluctuated between 50° and 70° F. On the 8th June two handfuls of earth from the pot were well mixed with about a litre of tap water, and a quantity of the mixture was then passed first through a Berkefeld and afterwards through a Chamberland F filter. The earth had a distinctly putrefactive odour. On the 9th June 40 cc. of the twice filtered liquid was injected under the skin in front of the near shoulder of a pony. At the same time another pony drank half a gallon of water made slightly muddy by the addition of some of the earth of the above-mentioned pot.

Both ponies remained apparently well and preserved normal temperatures during the following nine days, when the taking of their temperatures was discontinued. They were kept under observation till the 5th July, when, being apparently quite well, they were used for another experiment.

Experiment VI.—On the 7th December last a horse drank a quantity of water made muddy by the addition of two handfuls of

earth from the pot mentioned in the preceding experiment. Between the 6th July and 7th December the pot had stood in the laboratory, and at the latter date the earth had become hard, dry, and caked, and had lost its putrefactive odour. On the 8th, 9th, 10th, and 11th December the horse drank further quantities of water made muddy by the addition of earth from the pot. In all, the animal must have swallowed about a pint of earth.

The horse remained apparently well and had a normal temperature till the 21st December, when the temperature was found to have risen to 103.6° . On the 22nd it was 103° , on the 23rd 102.6° , and on the 24th 102° . On the 25th the temperature had again become normal, and the animal remained apparently well till the 25th February last, when it was inoculated with 5 cc. of preserved horse-sickness blood. It died from typical horse-sickness on the eleventh day after inoculation.

It does not appear to be possible to say with certainty what was the cause of the rise of temperature from the 21st to the 24th December. It is perhaps most probable that the rise was unconnected with the ingestion of the earth to which horse-sickness blood had been added; but, on the other hand, neither the late period at which the disturbance of temperature occurred, nor the fact that the animal subsequently became infected when inoculated with virulent unfiltered blood, makes such a conclusion certain. Experiment III. shows that the period of incubation may be prolonged much beyond that which is ordinarily observed; and, as Edington has pointed out, an animal that has given evidence of infection after inoculating with a small dose of virulent material may succumb when it is inoculated a short time afterwards with a larger dose.

There is, however, no doubt about the interpretation which ought to be placed on the result of Experiment IV. It is evident from that experiment that a very advanced stage of putrefaction may be reached without the blood having thereby lost its infective property.

EFFECT OF INCUBATION AT THE BODY TEMPERATURE ON THE VIRUS.

Experiment VII.—On the 19th January 1900 a mixture of three volumes of tap water and one of blood from a pony dead of horse-sickness was filtered through a Berkefeld filter. Part of the filtrate was poured into a sterile tube, which was placed in an incubator at 37° C.

The above-mentioned filtrate was left in the incubator till the 6th March (forty-seven days), when the liquid was still quite clear, and showed no bacteria on microscopic examination. On this date 5 cc. of the liquid was injected in front of the near shoulder of a pony. The result of the experiment was entirely negative.

The experiment indicates that the agent of infection in horse-sickness loses its vitality with prolonged incubation at the body temperature. In another experiment positive results were obtained by inoculating with a similar liquid which had been left for the same time at the temperature of the laboratory.

Experiment VIII.—Although in previous experiments the various media inoculated with horse-sickness blood and afterwards incubated

had not developed any evidence of bacterial growth, it appeared to be possible that, at least in liquid media, some multiplication might take place without sensibly affecting the transparency of the medium, owing to the very minute size of the bacteria, and that the only reliable test of the success or failure of attempts to cultivate the virus artificially would therefore be experimental inoculation of the incubated medium to which a trace of horse-sickness blood or other infective material had been added. Accordingly a quantity of horse-sickness blood was diluted with six volumes of water and an equal quantity of blood serum from a healthy horse, and passed through a Berkefeld filter. This filtrate was infective, for a pony inoculated with 60 cc. of it died from horse-sickness. Part of the filtrate was decanted into a test-tube, and this was incubated for thirteen days at 37° C. From it a loopful of liquid was then transferred to a test-tube containing serum-bouillon (one of normal horse serum to two of ordinary peptone bouillon), which was incubated at the same temperature. At the end of four days a second serum-bouillon tube was inoculated from the first, and after five days' incubation it in turn was used to inoculate a third. All three serum-bouillon tubes were incubated till the 6th March 1900, on which date they were used to inoculate three ponies, 5 cc. of the serum-bouillon being injected under the skin of each animal. At the date of inoculation the first serum-bouillon tube of the series had been incubated for fourteen days, the second for ten days, and the third for five days. None of the three animals became infected.

INFLUENCE OF THE METHOD OF INFECTION ON THE PERIOD OF INCUBATION.

In the former article it was shown that when considerable quantities of virulent blood were administered by the mouth the experimental animals became infected after a period of incubation which did not differ from that which is observed in animals infected by subcutaneous inoculation. The following experiment was carried out in order to ascertain whether there is any difference in the length of the period of incubation when intravenous injection of the virulent material is the method of infection.

Experiment IX.—On the 5th July last two ponies of approximately the same size were each inoculated with 5 cc. of preserved citrated blood-plasma, the liquid being injected under the skin of the neck in the one case, and into the jugular vein in the other. The subsequent temperatures of the two animals is shown below.

<i>Intravenous.</i>				<i>Subcutaneous.</i>			
July	5	.	99'4	July	5	.	100
"	6	.	99	"	6	.	100'2
"	7	.	100	"	7	.	100'6
"	8	.	99'4	"	8	.	100'5
"	9	.	102	"	9	.	104
"	10	.	104	"	10	.	105
"	11	.	103	"	11	.	106'5
"	12	.	104'5	"	12	.	107
"	13	.	101'2	"	13	.	101'6

The pony inoculated subcutaneously died on the 13th July at 3.45 P.M., and the following are the notes of the *post-mortem* examination:—

Autopsy.—No froth or liquid in the nostrils. No lesion at the seat of inoculation. A little gelatinous œdema along the course of the trachea in the neck. No measurable liquid in the peritoneum. Peritoneum and abdominal lymphatic glands normal. Kidneys somewhat congested. Spleen normal in size, appearance, and firmness. Liver has some "milk spots" on its anterior surface, and it is full of blood. Stomach is moderately filled with food materials; the mucous membrane of the greater part of its right half is deeply congested, and shows a considerable number of hæmorrhages into its substance. The small intestine shows at a few places some congestion of its mucous membrane, and the large colon shows diffuse general congestion of its lining membrane. The cæcum is almost normal. The bladder contains about half-a-pint of turbid urine; its mucous membrane is normal. Urine distinctly albuminous. There is no measurable liquid in either pleural sac. The pericardium contains about 4 ounces of yellowish transparent watery liquid. Both lungs are markedly œdematous, and the trachea and large bronchi contain a considerable quantity of frothy spume. In the left ventricle there are numerous sub-endocardial hæmorrhages. The tongue and throat are normal.

The pony inoculated intravenously also died on the 13th July, at 0.45 P.M., and the lesions found in it were as follows:—

Autopsy.—A few ounces of watery liquid in the peritoneum. Serous membrane normal. Abdominal lymphatic glands normal. Spleen normal in size and firmness. Liver contains some calcified tubercles; its parenchyma is congested. Kidneys congested. The stomach is well-filled with food materials; the greater part of the mucous membrane of its right half is livid from congestion, and it shows some small ecchymoses. Patches of congestion throughout the small intestine. Livid congestion, not quite uniformly distributed, of the mucous membrane of the large colon and cæcum. The bladder contains about 4 ounces of turbid urine, which is slightly albuminous (cold nitric acid test). The pleural sacs contain about half-a-gallon of straw coloured, almost transparent watery liquid, and about 8 ounces of similar fluid is present in the pericardium. The lungs are in a condition of extreme œdema, and at numerous places the pulmonary plasma is raised into blister-like elevations by a yellowish gelatinous exudate. The trachea and bronchi contain a large quantity of fluid and froth. The heart is firmly contracted and normal. At its base there is a considerable quantity of gelatinous exudate around the trachea and great vessels. The tongue and throat are normal.

The nature of the exudate seen at the heart from what might have been expected, but one might have anticipated that as what appears to be a serous membrane exists there, so if inoculation would have been done intracardially, the exudate at the heart would have been

REPORT OF A POST-MORTEM EXAMINATION.

The following was written by Mr. J. H. B. on the 13th July 1891, after the post-mortem examination of a horse which died on the 12th July 1891, after being inoculated with the virus of the disease on the 11th July 1891.

observe, however, that M. Nocard, in an article which appeared during the course of the present year,¹ has arrived at the conclusion that the virus of horse-sickness is arrested by a Chamberland filter, and, as this is a view in conflict with the results of a number of the experiments previously recorded by me, the question appears to demand re-examination.

In the first place, it may be observed that the article which appeared in this *Journal* twelve months ago seems to have escaped M. Nocard's notice. At least that inference appears to be justified from the fact that as recently as the month of January last he described to the Société Centrale de Médecine Vétérinaire an experiment proving that the virus of horse-sickness is not arrested by a Berkefeld filter, without mentioning that this had been established by other experiments previously published.

M. Nocard's conclusion that a Chamberland filter suffices to arrest the virus of horse-sickness appears to be founded on the negative result of a single experiment, in which he inoculated a pony with 40 cc. of a 3 per cent. mixture of pericardial liquid and Seine water which had been passed through such a filter. An exactly parallel experiment in which the liquid used for inoculation had been passed through a Berkefeld had a positive result. I cannot pretend to be able to explain the discrepancy between the result of this experiment and the results of the five experiments with Chamberland filters described in my previous article. I shall content myself with leading further evidence to show that the result obtained by M. Nocard constitutes an exception, and that the ordinary Chamberland filter, apparently free from any accidental defect, may allow the agent of infection of horse-sickness to pass through it.

Experiment X.—On the 14th July last a quantity of mixed pleural and pericardial liquids taken in an unsterilised vessel, without any precautions, from the chest of a pony dead of horse-sickness on the previous day (faintly tinged with blood, which escaped from the divided vessels in the chest wall) was passed first through a Berkefeld and afterwards through a Chamberland B filter. In both cases the filtration was carried out under pressure, the manometer attached to the flask indicating a pressure of about 26-28 inches of mercury. The absence of any fissure or other gross defect from the second filter was indicated by the fact that even under the pressure mentioned the watery liquid, already deprived of any visible particles by the Berkefeld filter, passed very slowly, only 25 cc. of filtrate being obtained in two hours. In this and all the following experiments the filter, flask, and fittings had been sterilised in an autoclave at 120° C., and the syringe used for inoculation was boiled in water immediately before use.

A pony was inoculated on the right side of the neck with 18 cc. of the above-mentioned twice-filtered liquid (14th July). The record of the animal's temperature afterwards is shown below.

July 14 . . .	101	July 19 . . .	104.2
" 15 . . .	100.8	" 20 . . .	105
" 16 . . .	101	" 21 . . .	103
" 17 . . .	101.4	" 22 . . .	101
" 18 . . .	102		

¹ Recueil de Médecine Vétérinaire, January 1901.

The pony died on the 22nd July, and the following are the notes of the *post-mortem* examination.

Autopsy.—Peritoneum normal, and no liquid in the cavity. Abdominal lymphatic glands normal. The stomach contains a small quantity of food material and several worms (*A. meg.*). The mucous membrane of the right half is intensely congested, being for the most part of a port wine colour. Throughout the small intestine there are patches of congestion and numerous petechial hæmorrhages. There are also some patches of congestion in the cæcum and large colon. Numerous *A. meg.* in the small bowel. Spleen, liver, and kidneys normal. The bladder contains a few ounces of clear urine, which gives no albuminous reaction with nitric acid. Mucous membrane of the bladder normal. There is no measurable liquid in the pleural sacs. The pericardial sac contains two ounces of clear, watery, straw-coloured liquid. The lungs are quite normal in appearance, but there is a small quantity of gelatinous exudate around the end of the trachea. Numerous petechial hæmorrhages are present under the endocardium.

One of the pharyngeal glands is a little enlarged, and contains a small quantity of pus, which shows streptococcus chains. There is no lesion at the seat of inoculation, but on both sides of the neck, and in the region of the withers, there is some clear jelly-like exudate in the inter-muscular connective tissue (none sub-cutaneous).

In the absence of any lesion at the point of inoculation, there is no reason to suppose that the presence of pus in the pharyngeal gland had any connection with the experiment.

Experiment XI.—On the 21st July 1900 a quantity of citrated blood-plasma, taken from a horse-sickness subject on the 28th February previous, was mixed with nine volumes of bouillon, and filtered through a Chamberland B filter. The liquid passed very slowly under pressure, only 40 cc. being obtained in two hours. A pony was inoculated subcutaneously in front of the near shoulder with 15 cc. of the filtrate. The temperature of the animal afterwards is shown below.

July 21 . . .	101·2	July 26 . . .	101
„ 22 . . .	101	„ 27 . . .	101·4
„ 23 . . .	101·4	„ 28 . . .	105
„ 24 . . .	101	„ 29 . . .	104·4
„ 25 . . .	101·2	„ 30 . . .	101·2

The pony died on the evening of the 30th July, and the *post-mortem* examination was begun immediately.

Autopsy.—No lesion at the seat of inoculation. Peritoneum normal; no liquid in it. Stomach moderately filled with food materials. Mucous membrane of right half shows some livid congestion, but less than usual.

No measurable liquid in the pleural sacs. The pericardial sac contains about a quart of clear straw-coloured liquid. There are some ecchymoses under the endocardium in both ventricles. There is no notable œdema or congestion of the lungs, and there is no froth or liquid in the trachea or large bronchi.

Experiment XII.—On the 13th of September last a quantity of blood which had been taken after death from the jugular vein of the

subject of Experiment III. on the 16th August, and which was now found to have become partially putrid, was mixed with ten times its volume of tap water, and passed under pressure through a Chamberland F filter. On the same date 10 cc. of the filtrate was injected under the skin in front of the off shoulder. The animal's temperature afterwards is shown below.

		A.M.		P.M.
Sept. 13	. .	—	. .	100'2
" 14	. .	100	. .	100
" 15	. .	100'2	. .	100'6
" 16	. .	100	. .	100'2
" 17	. .	99'8	. .	100
" 18	. .	99'8	. .	100'4
" 19	. .	100'8	. .	102
" 20	. .	100'6	. .	103
" 21	. .	100'4	. .	103'4
" 22	. .	103	. .	104
" 23	. .	102'4	. .	104'8

The pony died on the 24th September at 4 A.M. The abdomen was eviscerated immediately to delay putrefaction, and the *post-mortem* examination was completed next forenoon.

Autopsy.—No lesion at the seat of inoculation. No liquid in the peritoneum. Right half of stomach intensely congested. Numerous patches of congestion in the small intestine, and a few in the large. Kidneys and liver congested. Spleen normal. Bladder contains about half a pint of slightly turbid urine, which on being tested with nitric acid gives a slight albuminous reaction. The pleural cavities contain about a pint of clear yellowish liquid with a very faint tinge of red. About three ounces of similar liquid in the pericardium. Both lungs intensely œdematous, with an abundant accumulation of a yellow gelatinous exudate under the pleura. The trachea and large bronchi contain a considerable quantity of thin yellowish liquid and froth. Heart normal. Some gelatinous exudate at the root of the lungs. Tongue and throat normal.

Experiment XIII.—On the 19th March last a quantity of horse-sickness blood diluted with about ten volumes of water was passed through a Berkefeld filter. On the following day a small quantity of putrid blood was added to the filtrate, which was then passed through a Chamberland B filter. The liquid passed very slowly under a pressure of from 26 to 28 inches of mercury, only about 80 cc. being obtained in five hours. A tube of bouillon was inoculated with a large loopful (platinum needle) of the liquid before it was passed through the second filter, while another tube was inoculated with a similar quantity of the twice filtered liquid, and 10 cc. of the latter was decanted into a sterile tube. The three tubes were then placed in an incubator at 37° C. On the following day the tube which had been inoculated from the liquid prior to filtration was turbid from bacterial growth, and had already a distinct scum on the surface, but the other two tubes remained sterile. The incubation of the tube containing the decanted filtrate was continued for four days, and a loopful of liquid from it was then used to inoculate a tube of bouillon, which remained sterile when incubated. These experiments

left no room for doubt as to the efficiency of the Chamberland filter for the arrest of ordinary bacteria.

On the 21st March 60 cc. of the above twice filtered liquid was injected under the skin in front of the left shoulder of a pony. The animal died on the 29th March. At no time had it shown any serious symptoms of illness, but on the two days preceding its death it had appeared rather dull, and on the last day the breathing was somewhat accelerated. The temperature of the pony after inoculation is shown below:—

		A.M.		P.M.
March	21	101·2		101
"	22	99·8		100
"	23	100		100·2
"	24	101·4		101·2
"	25	102		102·2
"	26	102		104·2
"	27	103		105
"	28	104		105

The following are the notes of the *post-mortem* examination:—

Autopsy.—No lesion at seat of inoculation. About 2 pints of straw-coloured watery liquid in the pleural sacs, and about 6 oz. of the same in the pericardial sac. Marked œdema of the lung tissue, associated with swelling of the interlobular septa, and at some places the accumulation of a yellow gelatinous material under and into the pleura. Trachea and large bronchi contain a considerable quantity of yellow watery liquid and froth. The whole of the right half of the stomach shows intense livid congestion of the mucous membrane, but the stomach itself is well filled with food materials. Throughout the last half of the small intestine there is a good deal of congestion of the mucous membrane, and some extravasated blood is mixed with the bowel contents. About 3 feet of the small intestine is at one place intussuscepted, but this portion shows not the slightest congestion of its peritoneal aspect. There is slight congestion of the mucous membrane of the large colon and cæcum. The spleen is about normal in size and appearance, but the liver and kidneys are very full of blood. Mesenteric and other abdominal glands normal. Tongue and throat normal.

Experiments VII. and VI.—On the 12th April last a pony was inoculated subcutaneously with 4 cc. of preserved horse-sickness blood, and the animal was killed by bleeding on the 20th April, when it was evidently near the point of death. Part of the blood was collected in a jar containing a solution of citrate of potash, the amount of the salt being sufficient to make a $\frac{1}{2}$ per cent. solution with the blood collected. Another part of the blood was collected without any admixture in a sterile jar. Both jars were set aside till the 23rd April, on which date a quantity of the upper plasma layer from the citrated blood was mixed with some serum from the second jar, in the proportion of two of plasma to three of serum. The mixture was passed through a Berkefeld filter, and on the 26th April a small quantity of normal blood was added to the clear filtrate. The filtrate was then divided, and part of each half was passed through two different Chamberland No. 5 filters. In each case the liquid passed

slowly under a pressure of about 26 inches of mercury, about 70 cc. being obtained in two hours from one of the filters, and about 50 cc. in three-quarters of an hour from the other. The efficiency of the two filters for the arrest of ordinary bacteria was tested exactly as in the preceding case. A tube of bouillon inoculated with a trace of the liquid before filtration rapidly became turbid and putrid on incubation, whereas tubes inoculated from the filtrates and the decanted portions (about 10 cc.) of the same remained quite sterile after several days' incubation.

On the 29th April the two separate filtrates were used for the inoculation of two ponies, 40 cc. of liquid being injected in front of the right shoulder in each case.

The following shows the temperatures of the two ponies after inoculation:—

<i>Chestnut Pony.</i>			<i>Brown Pony.</i>		
	A.M.	P.M.		A.M.	P.M.
April 29 .	100	—	April 29 .	100	—
" 30 .	99·8	100·2	" 30 .	99·6	100
May 1 .	99·4	98·4	May 1 .	99·4	99·8
" 2 .	98·6	99	" 2 .	98·6	99
" 3 .	99·6	100	" 3 .	99	99
" 4 .	101·2	102	" 4 .	101·6	102
" 5 .	102·8	103·6	" 5 .	103·8	104·4
" 6 .	102·8	103	" 6 .	103·6	104·8
" 7 .	97·8	—	" 7 .	102	104·6
			" 8 .	102·8	101·8

One of the animals died on the 7th May at 2 P.M., and the other on the 9th May at 1 A.M.

The abnormalities discovered at the autopsy in the two cases were almost identical, and it does not appear to be necessary to describe them at length. In neither case was there any appreciable reaction at the seat of inoculation in front of the shoulder, but in both there was some yellowish gelatinous œdema between the muscles and along the course of the trachea. In both animals there was also a quantity of clear watery liquid in the pleural and pericardial sacs, as well as intense livid congestion of the mucous membrane in the right half of the stomach.

Experiment XVII.—On the 1st June of this year, a quantity of preserved horse-sickness blood was diluted with four volumes of tap water, and some putrid blood was added to the mixture, which was then passed through a Chamberland B filter. The mixture passed very slowly, only about 20 cc. of filtrate being obtained in five hours under a pressure of 26 inches of mercury. Here again a bouillon tube inoculated from the filtrate, and a small quantity (about 5 cc.) of the latter decanted into a test tube, developed no evidence of bacterial growth when they were incubated, whereas a tube of bouillon inoculated with a trace of the liquid before its passage through the Chamberland filter soon became turbid when it was incubated.

On the same day (1st June, 15 cc. of the filtrate was injected in front of the right shoulder of a pony. Before it was made the subject of this experiment the animal was very thin and weak, and on the 5th June it was unable to rise without assistance. On the following day

it was unable to stand, and on that date 5 cc. of blood was drawn off from one of its jugular veins by means of a sterile syringe and immediately injected subcutaneously in front of the right shoulder of another pony.

The first pony was killed on the 7th June. Its temperature since the date of inoculation had been :—

June	1	.	.	.	.	99·8
"	2	.	.	.	.	100
"	3	.	.	.	.	100
"	4	.	.	.	.	99·8
"	5	.	.	.	.	100·2
"	6	.	.	.	.	100·6

Although the pony was killed on the sixth day after inoculation the *post-mortem* examination revealed the lesions which are most characteristic of horse-sickness, viz., some gelatinous œdema inside each shoulder, a quantity (about 5 ounces) of clear liquid in the pericardium, and intense, almost general, livid congestion of the right half of the gastric mucous membrane. There was no lesion at the point of inoculation.

The pony inoculated with blood from the above subject on the 6th June had afterwards the temperatures shown below :—

June	1	.	.	.	.	99·8
"	2	.	.	.	.	102
"	3	.	.	.	.	100·6
"	4	.	.	.	.	101·6
"	5	.	.	.	.	103
"	6	.	.	.	.	105·2
"	7	.	.	.	.	105
"	8	.	.	.	.	104·6
"	9	.	.	.	.	98·6

This animal died at noon on the 14th June, and the autopsy revealed the following abnormalities. Intense congestion of the right half of the stomach, associated with numerous small hæmorrhages into the mucous membrane; a quantity of jelly-like exudate along the course of the trachea in the neck; about 6 ounces of clear liquid in the pericardium, and a quart of similar liquid in the pleural sacs; slight œdema of the lungs; and patches of a gelatinous exudate under the pulmonary pleura. There was no visible reaction at the point of inoculation.

Including the cases described in my previous article, it will be found that in nine separate experiments animals have been infected with horse-sickness by inoculation with filtrates that had been passed through Chamberland filters. In three of these experiments the filter employed was of the F pattern, and in the remaining six the B pattern was used. It is important to note that four different B filters were used in the six experiments with that variety of the Chamberland apparatus, and that they were specially obtained from Paris for the purpose of the experiments, and bore the official stamp of the manufacturers. Until a larger series of experiments with different results has been published it is therefore permissible to maintain that, as a rule, neither the F nor the B form of the Chamberland filter is efficient for the arrest of the microbe of horse-sickness. It may even be suggested with some measure of

confidence that the limitation introduced into the previous sentence ("as a rule") might have been omitted, and that there is no exception to the rule stated. In saying this there is no intention to impugn the accuracy of the facts recorded by M. Nocard, but merely to question the soundness of the general conclusion which he has drawn from the result of a single experiment. It must be accepted as a fact that in M. Nocard's experiments 40 cc. of a liquid that had been passed through a Chamberland filter failed to infect a horse, whereas an identical quantity of the same material that had been passed through a Berkefeld sufficed to infect another animal, but this does not compel one to admit that none of the germs of horse-sickness passed through the particular Chamberland filter that was employed. It certainly appears to be possible that some of the germs did pass through that filter, and that the animal inoculated with the filtrate escaped infection because it was more resistant than the horse used for the parallel experiment with the Berkefeld filter, or because a proportion of the germs in the original liquid were arrested by the Chamberland filter.

In this connection it may be of interest to give the details of two experiments in which filtrates unexpectedly failed to infect the animals with horse-sickness.

Experiment XVII.—On the 14th May last, a pony was inoculated subcutaneously with 25 cc. of pericardial liquid which (on the 10th May) had been passed through a Chamberland B filter. The liquid had been taken from the pericardial sacs of the subjects of Experiments XIV. and XV. of this article. The result was entirely negative, the animal preserving a normal temperature and every appearance of health during the next three weeks. Unfortunately, the whole of the mixed pericardial liquids from the two ponies had been passed through the Chamberland filter, and it was therefore impossible to make a control experiment to ascertain whether the unfiltered liquid was also non-infective with the same dose. Assuming, however, that the unfiltered material would have proved infective, the result of the experiment with the filtrate was in harmony with the view that this particular Chamberland filter was impermeable for the microbe of horse-sickness, or that the subject of the experiment was immune against the disease. Subsequent experiments showed that neither of these explanations of the result was tenable, for it was the same filter that was afterwards employed in Experiment XVI., and the same pony which, as part of that experiment, was inoculated with blood on the 6th June, and died from horse-sickness on the 14th June.

Experiment XVIII.—On the 18th October last a quantity of preserved horse-sickness blood (taken from a pony on the 6th December 1899) was mixed with nine volumes of tap water and passed through a Berkefeld filter. On the same date 1 cc. of the filtrate was injected into the left jugular vein of a pony. The pony's temperature remained normal during the following month, and during that period it appeared quite well. The negative result of the experiment might be interpreted as indicating either that the preserved blood had entirely lost its virulence, or that the Berkefeld filter completely arrested the agents of infection. The former explanation was subsequently made untenable, for a horse which was inoculated on the 25th February 1901 with 5 cc. from the same sample of preserved blood died from typical horse-sickness on the 6th March. Does this

compel one to adopt the alternative explanation suggested above? The answer must be in the negative, for there is a third possible explanation—viz., that the filtrate failed to infect not because it contained none of the germs of horse-sickness, but because it did not contain the number necessary to ensure infection.

These experiments are mentioned here because they indicate that unless very considerable quantities of a filtrate have been used for inoculation the negative result of an experiment does not warrant the conclusion that none of the agents of infection have been able to pass through the filter. It is at least possible, if not probable, that even a Berkefeld filter arrests some of the germs of horse-sickness, and *a fortiori* it is possible that although the liquid which has passed through a Chamberland filter is still found to be infective it has nevertheless been robbed of some of its contained bacteria.

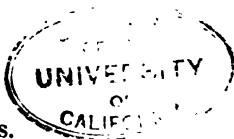
SOME REMARKS ON THE INHERITANCE OF ACQUIRED IMMUNITY.¹

By GEORGE OGILVIE, B.Sc., M.B., Edinburgh, M.R.C.P., London,
Physician to the French Hospital, and to the Hospital for
Epilepsy and Paralysis, Regent's Park, London.

IF our notions regarding the process of generation have become somewhat more distinct by recent investigations into the minute structure of the cell and the mode of its propagation, yet this advancement of our knowledge is so infinitesimal compared with the mystery in which the whole matter is enshrouded that it throws little or no light on the many problems of importance with which physiology and clinical medicine confront us concerning the laws of inheritance. Even with regard to that fundamental and much-disputed question of the transmissibility of acquired characters we have not come to anything like a general and complete understanding. Weismann's theoretical objection to the possibility of such transmission is derived from our inability to conceive how an alteration in the cells of the body could react upon the germ cells in such a manner as to induce in the latter a reproduction of the acquired character. It has to be admitted that the hypothetical and often fantastical constructions which have been devised to explain such a relation between body cells and germ cells are altogether unsatisfactory, and never rise above the level of more or less acceptable hypotheses. But in a subject so involved in obscurity the present incomprehensibility of certain relations can hardly serve as an argument against their existence. The development of the apparently uniform germ plasma into the infinite differentiation of a complex cell state is, although no longer a matter of doubt, perhaps not less inconceivable.

Of more convincing power is the critical part of Weismann's argument. It is his incontestable merit not only to have given a more precise shape and formulation to many questions relating to the subject of hereditary transmission, but also to have done away

¹ Reprinted from the *British Medical Journal*, 4th May, 1901.



with many prejudices by showing the frailty of the basis upon which they were founded. Frequently the hereditary transmission of some pathological condition, for instance, had been accepted, for no better reason than that it was found in two or more subsequent generations. If the weakness of such a conclusion is obvious it is nevertheless frequently to be met with in medical literature, and it is in this sense that only lately the complaint has been made, not without justification, that in discussing the hereditary transmission of pathological conditions, the question itself was frequently but incompletely understood. If Weismann has not succeeded in disproving the possibility of the hereditary transmission of acquired characters, his opponents are equally at a loss to furnish exact proof of its existence.¹

This fact is well illustrated by O. Hertwig's² excellent work on the cells and tissues, in which the whole theory of biogenesis and the problem of inheritance form the subject of a thorough and most exhaustive investigation. With many of the most distinguished biologists of our days, he believes that acquired characters are transferable to the germ. He endeavours to render this process intelligible by pointing to the analogy between it and certain properties of the brain substance, at the same time admitting that "this analogy is far from being an identity." But the proofs which he adduces in support of the existence of the supposed relation between body cells and germ cells are scanty and unconvincing. Two of them are taken from one-celled animals. The first is derived from the phototaxis of certain algæ, the second from certain "physiological varieties" of some bacteria, which under certain artificial conditions lose their toxic qualities, and transmit this "acquired character" of lost virulence to the next generation.

With regard to the irritability of the algæ to light, we are told that it can be modified by exposure to strong light and to high temperature, and that "nobody would be surprised" if their progeny also showed "some similar property." This supposition may be psychologically correct, but it does not furnish an example of the hereditary transmission of an acquired character. In the case of the bacteria the loss of their toxic properties can hardly be described as the acquisition of a new character. But, quite apart from the inadequacy of the testimonies thus adduced, it is obvious that observations taken from unicellular organisms are unsuitable to explain the mechanism by which the different cells of a complex body act and react upon each other.

As regards the more complex organisms Hertwig gives only one instance. This is taken from the domain of pathology—namely, the hereditary transmission of acquired immunity, which, as he says, has

¹ It goes without saying that I am not speaking here of the hereditary transmission of infectious diseases. Wherever this has been adduced to support the doctrine of the inheritance of acquired characters, it has been done in utter misconception of its meaning and scope. It has also to be remarked that bacillary infection of the germ plasma as the cause of congenital infectious disease, although perhaps a clinical postulate, has not yet been demonstrated pathologically. The experiments made by Baumgarten and Maffucci to prove germinal infection, by injecting tubercle bacilli into fowls' eggs, and thus producing chickens affected with tuberculosis, are based upon the same fallacy of which Sir John Falstaff was guilty when he objected to "pullet-sperm in his brewage." The hen's egg (ovum) is not the female germ (ovum), but an intermediate stage between the latter and, as Cressida has it, the "chicken i' the shell." The argument which underlies those experiments reads like a kind of pathological quibble on the double *entente* of the term "ovum."

Hertwig, Oscar, *Die Zelle und die Gewebe*, Jena, 1898.

been demonstrated by the experiments of Tizzoni and Ehrlich. But while the experiments of Tizzoni are inconclusive, those of Ehrlich point just in the opposite direction. Ehrlich's ingenious experiments,—made on mice with the vegetable toxins of ricin, abrin, and robin—have led him to the conclusion that no trace of immunity is ever conferred by parent upon offspring through the germinal cells, either by the sperm or by the ovum. This has been confirmed for the toxin of tetanus by experiments made by Hübener and Vaillard on mice, rabbits, and guinea-pigs, and for the toxin of diphtheria by experiments made by Wernicke on guinea-pigs. I am, therefore, unable to understand how Ehrlich's experiments could have been adduced in proof of the germinal transmission of immunity. Tizzoni, on the other hand, believed he had demonstrated the paternal—that is, germinal—transmission of immunity by experiments made on rabbits with the toxin of rabies.

These experiments exhibit a marked source of error, because the female animals were not in a normal condition, but had been previously rendered highly immune to the tetanus toxin. Roux and Calmette have shown that the artificial immunity to certain toxins is not strictly specific, that in the same way specific antitoxins may be protective against more than one toxin; thus, for instance, the tetanus antitoxin is also protective against snake poison, and the same property belongs to the serum of animals which have been rendered immune to rabies. The possibility is, therefore, not excluded that the serum of animals rendered highly immune to tetanus may have a certain influence upon the poison of rabies—in fact, that the immunity observed in the offspring of Tizzoni's experiments may not have been due to paternal transmission, but to the conveyance of the tetanus antitoxin from mother to child by the placental circulation. Moreover, Tizzoni's experiments have never been corroborated by any other observer. The germinal transmission of immunity, therefore, so far from being a fact established by experimental research, is negated by all the reliable experimental evidence we possess. It is also negated by all clinical experience. It is certainly a noteworthy fact that it is the only instance which a biologist of greatest learning is able to adduce in favour of the hereditary transmission of acquired characters.

The above-mentioned investigations by Ehrlich have also led to a positive result of considerable importance—namely, that the offspring may derive immunity from its mother in a twofold manner, by either the blood or the milk—that is, by the placental circulation or by suckling. It goes without saying that in these cases it is not the immunity which passes from the maternal organism to that of the child, but the antitoxin, and that the passive immunity of the offspring thus produced cannot be considered as an acquired character transmitted by true inheritance.

Ehrlich's results were obtained by experiments made on mice with vegetable poisons. Subsequent investigations have shown that these two forms of passive immunisation of the offspring do not necessarily go together. According to Vaillard, neither in guinea-pigs nor in rabbits does any degree of immunity to tetanus pass from mother to child by suckling; and Wernicke found that the suckling immunity to diphtheria in guinea-pigs, if it exists at all, is much less pronounced

than, for instance, the suckling immunity to tetanus in mice. Yet in rabbits and in guinea-pigs as well as in mice a certain degree of blood or placental immunity is conferred by the mother on the child with regard to both the tetanus and the diphtheria toxin. These facts, interesting in themselves from a biological point of view, impart at the same time a wholesome warning against rash and premature generalisations. In particular they seem to dissuade us from the unhesitating application of results gained by experiments on lower animals to human pathology. Ehrlich's experiments have of late been made use of to explain all sorts of real or imaginary phenomena of clinical medicine. In truth, however, up to the present day no clinical observations exist pointing to the existence of a similar transmission of immunity in the human race. If there is nothing to prove that immunity as an acquired character—that is, active or histogenic immunity—is ever transmitted to the next generation by true inheritance—that is, by the germinal cells, the placental and the suckling immunity also have, as far as we know, no exact parallel in human pathology.

Only a short time ago a question closely connected with this subject was again raised in one of our leading journals, and led to some controversial correspondence—the question of intrauterine vaccination. It seems to have escaped the attention of those who originated and took part in this discussion that exactly the same question had been raised for the first time some twenty-five years ago in the same journal, and that it has since been the object of much careful clinical research. All the most reliable evidence we have got on this point leads to a negative result. It will be found that the few apparently positive results which have been reported are more or less open to doubt, because the methods employed and the precautions taken afford no sufficient guarantee for their conclusiveness. Even if these exceptionable and more or less contestable cases are granted, the fact remains that, as a general rule, intrauterine vaccination does not take place. If vaccination is only a form of bacterial infection, it is an open question whether the few alleged exceptions are cases of intrauterine infection in its proper sense or of intrauterine intoxication—that is, of conveyance of the toxic agent of vaccination from mother to child through the placental circulation. Of the milk, as the vehicle for the conveyance of vaccinia, nothing is known.

It has long been remarked by clinical observers that the new-born enjoy a certain degree of immunity from certain acute infectious diseases which childhood is particularly liable to, such as measles scarlet fever, etc., and that this relative immunity extends over the first six months, and to a lesser degree even over the first year of life. This has been explained in different ways. While some are inclined to attribute it solely to the relative want of the opportunity for infection, which it is said is not given to its full extent in infancy, others have ascribed it to a certain state of congenital immunity to a peculiar condition of the infantile tissues.¹ At the same time it

¹ The remarkable experience which J. Schreibern ("Deut. med. Woch.", 1891, No. 8) has had with tuberculin in new-born children of non-tuberculous extraction—he injected forty healthy infants with large doses of tuberculin (up to 5 cc.) without any febrile or other effect—either seems to prove that the infantile tissues possess a degree of irritability to certain bacterial products which totally differs from that of the adult, or that the power of excretion is much higher. In the same way the tolerance which infants show to comparatively large doses of mercury may be explained.

has been observed that mothers suffering from such infectious diseases (also from smallpox) may on the whole suckle their healthy-born children without infecting them. This fact also has found different explanations. On the one hand it has been attributed to the natural immunity of the new-born in general to certain infections, and on the other hand to a particular immunity acquired either *in utero* or during lactation, and consequent upon the mother's disease. Here, again, Ehrlich's experiments have been referred to as a parallel. But it is evident that these experiments have been totally misunderstood. In Ehrlich's experiments the mother is in a high state of immunity, while in the case we are discussing the mother is suffering from the actual disease.

Exactly the same mistake has been made as I have pointed out before,¹ with regard to the explanation of the so-called "law of Profeta." Whatever the right interpretation of the infantile immunity may be in these different cases, there is no doubt that Ehrlich's experiments give no clue to it, nor that it can ever be regarded as due to hereditary transmission.

In fact, everything that is acquired by the child during its intra-uterine life cannot be considered as the outcome of heredity in its proper sense, even if due to a similar or the same condition in the mother. In this stricter sense, those characters only are inherited which lie "performed" in the germinal cells at the time of impregnation. By some writers on hereditary syphilis a difference has been made between hereditary and congenital syphilis, in so far as the latter term has been applied to the disease if present at the time of birth, while the former is restricted to those forms of the disease which do not lead to obvious symptoms until some time after birth. This distinction is unsatisfactory, and has generally been abandoned, because the syphilitic infection, whether germinal or placental, does not as a rule manifest itself until some time after birth, and because congenital syphilis in this restricted sense may follow either mode of infection. But a distinction has always, and rightly, been made between germinal infection on the one hand, and post-conceptional or placental transmission of syphilis on the other. Although we do not know by any difference in the outward symptoms whether the infantile syphilis is derived in one way or the other, yet from a pathological point of view this distinction is of considerable importance. Of course all paternal infection can only be germinal.² That such a thing as germinal transmission exists in maternal infection we can only presume from analogy, because, as said before, germinal and placental syphilitic infection do not differ in the nature or succession of the clinical symptoms they produce.

It seems that in the case of small-pox congenital immunity to vaccination in the child is occasionally produced where the mother is

¹ "British Journal of Dermatology," Vol. IX., 1897, p. 436.

² Indeed, the absolute proof of the possibility of the inheritance of acquired immunity should primarily be sought for on the paternal side. In this way all questions of mere evanescent immunity acquired from the mother *in utero* or during suckling, and which is to be regarded as nothing more or less than an accidental poisoning of the fetus or child, would be entirely eliminated. No believer in the doctrine either of "acquired characters" or of "acquired immunity" would be likely to assert that the dominant factor was the ovum rather than the sperm. If we assume that the sperm equally well with the ovum can "pass on" an "acquired character" or an "acquired immunity" to the offspring, the convincing proof of the hypothesis were best sought for through the father, because, unlike the mother, his part in the production of life ceases with the act of coitus.

suffering from the disease during gestation. A number of observations exist in which children born under such circumstances have been vaccinated without result. Statements of this kind are always open to the objection that the inefficiency of the vaccination was due to some imperfection of the method employed, or that vaccination was not tried repeatedly. Yet there are cases on record which seem to hold out against all reasonable criticism. But we do not know of any case in which the parent's disease or vaccination previous to impregnation has produced the same effect. It has been alleged that such an influence of the vaccination of previous generations could be traced in the fact that in countries in which compulsory vaccination is practised occasional small epidemics of variola are of a milder character than formerly. But this fact admits of a different and more natural interpretation. This mitigation is certainly partly due to the bulwark which general vaccination has erected against the spread of the disease, partly to the advancement in the treatment of infectious diseases in general, partly and not least to the more hygienic methods—such as prompt and complete isolation, etc. On the other hand, cases of a very severe, destructive, fatal character are not altogether uncommon even under these circumstances. As far as our present experience goes, no influence of the parent's vaccination previous to impregnation is traceable in the offspring, either by insusceptibility or by a diminished susceptibility to vaccination—that is to say, that nothing is known about a germinal transmission of immunity from parents who, previous to impregnation, had been subject to vaccination or to an attack of small-pox.

Ricord believed that a person who by a first attack of syphilis has acquired immunity to subsequent infection confers this immunity upon his offspring. But his frequently quoted saying "*C'est un heritage comme un autre*," is only one of his many *bon mots* which in course of time, and through the prestige of his great authority, have gained the credit of scientific truths. In my paper on *Congenital Immunity to Syphilis* I endeavoured to show that the facts which might be, and have been, adduced in its support are insufficient in number as well as in demonstrative power, and that, on the other hand, the general experience of every-day practice points in the opposite direction. What is true for these two infections holds good for all of them. It seems that in some infections, as for instance tuberculosis, parental disease creates in the germinal cells a certain predisposition which renders the offspring more liable to acquisition of the same disease, but we know of no infectious disease which in the same manner exercises a protective influence upon the product of generation. With regard to that congenital immunity which in some infectious diseases is occasionally conferred upon the offspring by a mother suffering from the disease during gestation, I have only to add that this immunity is always only temporary and of short duration, and that for this reason alone it could not be regarded as an acquired character.

If no clinical proof exists to show that parental immunity is ever transmitted by the germinal cells to the next generation, there still remains the possibility that such transmission takes place all the same, only to such a minute extent that it escapes detection by the rough and somewhat clumsy methods of clinical observations. In his

lectures on the Pedigree of Disease, Mr Hutchinson expresses views which are, on the whole, adverse to the hereditary transmission of acquired immunity, yet he cautiously adds that if it exists at all it can only be in an "infinitesimal" degree. If I understand him rightly, he means to leave open the possibility that during the succession of generations each succeeding generation may acquire an additional degree of immunity, and that this originally infinitesimal amount may accumulate to a finite appreciable amount, or even to total immunity, in the same way that Darwin, to account for the hereditary transmission of acquired characters, presupposes the accumulative effect of a long series of generations during which the influence which first led to the acquisition of the new character was constantly at work. In this form the theory of the hereditary transmission of immunity has indeed frequently been advanced in order to explain the immunity which certain human races—negroes and mulattos—enjoy with regard to certain tropical diseases. While originally liable to the deleterious influence of these infections in the same way and to the same extent as the white races, they are said to have become proof against them by living in regions in which every following generation is exposed to and attacked by the same poison, and in which every generation leaves to the next the "infinitesimal" amount of immunity inherited from the preceding *plus* the equal amount acquired by itself. With regard to this theory I have in the first place to remark that it is by no means identical with that first advanced by Dr W. C. Wells as far back as 1813, and adopted by Darwin, which explains this racial immunity by natural selection, by the "survival of the fittest." According to Wells, among the different varieties of men which populated the middle regions of Africa some were better able to resist the endemic maladies than others, they consequently survived and spread, while the others perished. It, therefore, is not an acquired character that was transmitted, but a natural immunity inherent in a dark-coloured race. This theory is unsatisfactory, because, while it may explain the extermination of the light-coloured races and the survival of the others, it does not account for the immunity of the latter. Not long ago, the same hypothesis has been extended to the microcosm of the animal cell state, and has been made use of to explain acquired immunity in general (Grawitz)¹. The "fittest" of the cells, those best able to resist the toxic influences of bacterial poisons, survive and multiply, while those of inferior powers of resistance succumb. But in this extension the theory is less satisfactory still, because there is no imaginable reason why of an aggregate of uniform cells, without any "racial" differences whatever, some should be endowed with defensive properties which the others want. Any theory which explains the immunity of certain races by some natural process of evolution will be more acceptable than this "first case of recognition of the principle of general selection," which leaves the racial immunity altogether unexplained as a mysterious appendix of a pigmented skin surface. But this accumulation hypothesis, which implies the co-operation of inherited and acquired immunity, if at first sight plausible and captivating, is also not free from objections.

In the first place a more thorough study of tropical diseases in our colonies has somewhat shaken the unconditional belief in many a

¹ "Virchow Archiv.," Bd. CXII., 1888.

statement which by text-books has been stamped with an almost axiomatic and catholic character. The immunity enjoyed by coloured races seems by no means so absolute as has been generally taught. In not a few cases on close enquiry—and a reliable previous history is, under the circumstances, not always an easy matter—the immunity of the adult natives seems to have been the result of an attack in early life. On the other hand, it is the newcomer, the inexperienced settler, who is most exposed to infection, while those who become accustomed to the climate and a suitable mode of living are much less in danger. But there is one circumstance in particular which more than any other detracts from the plausibility of this theory, that is, that no similar gradual immunisation has ever been observed in the white races with regard to any of the infectious diseases which they have suffered from.

With regard to vaccination it may be objected that a long enough time has not yet elapsed to produce such a result. The same objection, however, could hardly be raised in the case of syphilis, or indeed that of smallpox, unless one assumes that the struggle between the black races and certain tropical diseases dates back to prehistoric times. Until we have at least some clinical or experimental proof that infectious diseases lead by themselves to a gradually increasing immunity in the offspring, the accumulation theory as an explanation of racial immunity will remain an unsubstantial hypothesis.

FOOT-AND-MOUTH DISEASE—SYMPTOMS AND LESIONS.

By ARTHUR H. BERRY, M.R.C.V.S., London.

FOR purposes of description the symptoms of foot-and-mouth disease may be conveniently divided into constitutional and local, and, as the symptoms to a greater or lesser degree are dependent on the lesions, and the presence of the lesions themselves are a *sine qua non* to a prompt and accurate diagnosis, it would appear that the semiology of the disease is incomplete without some reference to the nature and course of the lesions, both being closely related to each other and of great value to the clinician.

SYMPTOMS AND LESIONS IN CATTLE.

The invasion of the virus into the animal body after an incubative period of very short duration is followed by symptoms sudden in appearance, and denoting septic disturbance. The general premonitory symptoms are: isolation, if at pasture, of one or more animals attacked; the desire to seek shelter and warmth; dulness and depression; falling off of the appetite, and in those cases in which the internal temperature is high (105-108° F.) the refusal of food; rumination is partially or entirely suspended; and the animal grinds its teeth, has a staring coat, is hide-bound, and tucked up in appearance.

When milch cows are affected the milk supply is speedily diminished.

There is an increase in the pulse and respirations, particularly in

cattle suffering with acute lameness arising from the pain of foot lesions, and the animal is appreciably distressed.

In the early stages the bowels are constipated, the fæces hard and dry, varying in firmness and dryness according to the degree of rise in temperature. The constipation soon disappears with the fall of the internal temperature, and, in the latter stages, a foetid diarrhoea is more often present; the back is arched, the animal soon appears hollow or empty; in fact, the loss in condition is well marked, rapid, and surprising, especially noticeable in the case of fat stock.

The extremities (seats of foot lesions excepted) are cold, there is shivering of the body or twitching of the muscles under the skin, the head is held down, the ears frequently droop, the eyelids are semi-closed, the animal appears drowsy, and the breath is foetid. Swelling of the muzzle and lips, at times extending over the nasal bones, catarrhal discharge from one or both nostrils and eyes (with tendency to accumulate around the interior of the nostrils and inner canthi of eyes) is more or less constant, especially so in the case of young store cattle which are subject to exposure in open yards, bleak pastures, or sea voyages.

From observations made during recent outbreaks of this disease in sheds and byres a very early symptom of interest—if not of value—may be detected in the sucking of the lips and frequent deglutition of saliva, which, in the course of the next twenty-four hours, is manifested by the characteristic, typical, and valuable clinical symptom of the disease—"smacking of the lips."

The "smacking of the lips" is a symptom present in a very high percentage of animals affected; in fact, a recent case would be most conspicuous without it. During the existence of this symptom it can be distinctly heard at a distance of many yards. It occasionally happens (when arriving on the premises where the disease is suspected to exist, if only one or two animals are attacked) that the "smacking of the lips" is not heard; the absence of this does not, however, materially lessen the value of so important a clinical indication of this disease, for, soon after the vesicles have ruptured, this symptom disappears, or, if heard, is only at irregular intervals.

The "smacking of the lips" is accompanied by excessive salivation, the quantity varying in individual cases, but being usually copious. The saliva at first accumulates round the lips in a frothy condition (which condition appears essentially dependent on the champing motion of the jaws) and drops from the mouth in small froth-like masses; or when animals are in the open it may be carried by the wind some distance before reaching, in many instances, not its final, but its intermediary destination—the ground—to affect, if opportunity occurs, other animals.

As the disease advances the saliva rather dribbles from the mouth, especially the commissures, in long threads—ropy saliva.

An inflammatory condition of the pharynx, larynx, tracheal and bronchial tubes is often associated with the disease, and a painful cough is present; in these cases rumination is attempted with difficulty, and often is delayed.

During the progress of the disease the temperature subsides, and, providing the disease is running an uncomplicated course, the appetite is gradually restored, and rumination is resumed.

Food, at first, is taken into the mouth very cautiously, particularly with long coarse food stuffs ; when in the mouth it is rolled from side to side, and often, during attempts to masticate, is dropped out, for, although the animal is willing to feed, if the lesions are extensive, it is unable, owing to the soreness of the mouth, which is only increased by the presence of food.

Animals left at pasture become very low in condition and emaciated. Firstly, owing to their inability to use the organ of prehension, and, secondly, on account of acute lameness, they are unable to search for food. In advanced cases they may be seen resting upon the sternum, or dragging themselves about the ground.

Diagnosis from the symptoms exhibited and the nature of the lesions presented, may be confirmed by the almost constant evidence of contagion amongst the stock, as also by the suddenness with which this malady appears without previous history.

In cases in which mouth lesions appear (almost constant in bovine species) a vesicular formation may be observed affecting the mucous membrane of the mouth, the principal seats being, inside of lower lip, inside of upper lip, chiefly at or about the junction of the lip with pad, on the pad, immediately behind the pad, at any position along the surface of the hard palate, but usually towards the posterior portion, less frequently the mucous membrane covering the inside of the cheeks, and, occasionally, at the line of demarcation between upper or lower lip with the skin proper.

The vesicles vary in size from that of a large pin's head or pea to the size of a walnut or even larger, causing displacement of the affected lip ; in other cases several small vesicles in close apposition coalesce, forming one of large size, which, when ruptured, leaves a large circular or oval bright red base.

A true vesicle, whether large or small, is an elevation of mucous membrane or skin (epithelium), corresponding to a blister or bladder formed by thermal or chemical action, and the vesicle is rich or sparing in a lymph-like exudate forming its contents, or "vesicular matter." This matter is charged with the highly virulent *contagium* of the disease.

The exudate is for the most part a thin, semi-transparent, light straw-coloured, or almost colourless fluid, and when the vesicle is ruptured by the fingers or has burst spontaneously it escapes, exposing to view a bright red or scarlet base and a ragged edge of loose epithelium.

The loose portion of epithelium soon becomes necrosed, forming a slough over the lesion, and it is assisted in keeping its position by coagulation of part of the exudate ; this peels off, or in a short time is rubbed off by the movements of the tongue, hastened by the presence of coarse food in the mouth.

The bright redness, however, soon disappears, the base becoming covered with a thin, opaque, lymph-like coating, which imparts a light yellowish-brown colour to the part.

There is under ordinary circumstances no tendency to suppuration, and, providing the lesion is not extensive, repair soon takes place, the epithelium growing from the periphery to the centre of the wound.

Granulation of the part is extremely rare, and then due to the

presence of some persistent irritant, complicating the course of the disease.

The tissue of the tongue adjacent to the lesions remains to the naked eye quite normal in appearance and structure; an inflammatory (chronic) or cirrhotic condition of this organ is but rarely present, and then rather as a sequel to the disease.

The ruptured vesicle heals as an ordinary superficial wound.

Other common seats of vesicular formation are the anterior surface of the muzzle and immediately within the interior of the nostrils, where they may be large in size (interfering with respiration), are of a glossy, semi-transparent appearance, and shortly after rupturing leave a yellowish loosely attached slough.

Aggravated by the action of the excoriating catarrhal discharges, the muzzle may become completely denuded of its skin, exposing a large, intensely hyperæmic surface.

The presence of vesicles on the tongue can be best detected by having the animal's head held well up towards the light, as during examination of this organ a good natural light is specially required to discover their presence, if small.

Some vesicles are not larger than a pea, slightly elevated from the surrounding surface, and only denoted by the ruffled appearance of small isolated portions of mucous membrane.

In many cases these non-ruptured small vesicles (as also larger ones) are very sparing in exudate, and during an ordinary examination, particularly if the light is not good and the animal is restless, may pass unobserved.

The small elevations can be felt by gently passing the finger over the suspected spot; pressure applied will not always tend to rupture them, but if these undesirable portions of mucous membrane are taken firmly between the finger and thumb and slight tension applied they burst, revealing a small bright red spot (base). On the other hand, vesicles on the tongue may be of very large size, from that of a shilling to that of a five-shilling piece, or even larger, and contain large quantities of exudate; these rupture quickly and easily, often during manipulation of the tongue, leaving the typical scarlet red base and a large piece of mucous membrane hanging from the tongue. The lesions are, for the most part, circular in shape, but occasionally oval or irregular. Vesicles of various size are often found on the tip and sides of the tongue, extending to its under surface, and when manipulating the tongue to withdraw it one must not omit to examine the part which the hand or finger is covering.

Vesicles may form on any part of this organ, but the middle and lower or anterior portion is the more constant. It is very uncommon to find independent or isolated vesicles situated on the under surface of the tongue, although extension of lesions from the edges may be present.

In extensive formation of vesicles the lower fourth of the tongue may be one large, angry-looking, painful sore, the mucous membrane having peeled entirely off, or hanging out of the mouth from the tip or side of the tongue. In rare and severe cases of extensive lesion, associated with much swelling, the tongue is held firmly between the incisors and the pad, and occasionally the tip drops off.

Vesicles on the pad are single, multiple, or confluent, and some-

times almost the entire surface of the pad is involved, having a raised, thickened appearance, the epithelium separating in one large crust.

The exposed base of this lesion, however, is but very slightly, if at all, tinted with colour, and soon after the epithelium has been cast the affected part can scarcely be distinguished from the surrounding healthy membrane. No definite or hard and fast rule can be laid down, but after from a day to a week, varying according to the size of the lesion affecting the tongue, the base has become covered by a thin layer of smooth epithelium, appearing below the level of the surrounding mucous membrane. In the average case, after resolution of the part is complete, little or no cicatrix is present, and it would be difficult to point out the situation of the pre-existing lesion. In more extensive lesions of the tongue, requiring several weeks before complete resolution of the part, the epithelium can be seen converging towards the centre of the wound, and the papillæ can be detected disseminated on the surface of the lesion itself.

Lesions on the buccal membrane after repair can be seen as circular or oval well-defined delicate pink patches, which in the course of a week or so become normal in appearance.

In concluding the remarks respecting the lesions of the mouth, I have observed that manipulation of the tongue is frequently followed by "smacking of the lips," which is of clinical value in an apparently undeveloped case (although its presence has not been detected prior to examination), and in all cases of a doubtful nature the thermometer is of the greatest value if the disease is suspected but not manifested. Excessive resistance on the part of the animal to examination of the mouth is often a suspicious indication of the presence of lesions.

Foot lesions may or may not be present. In some outbreaks mouth lesions predominate, but in most instances where there are several cattle affected lesions in both foot and mouth will be present, and often individual animals are affected in mouth, foot, and in the case of milch cows, the udder; but, generally speaking, mouth lesions are most constant and extensive in cattle.

In the average case foot lesions are comparatively slight in extent, although frequently from the manifestation of lameness they would appear to be most severe; this can be clearly understood when we consider, not only how highly vascular and sensitive are the structures implicated, but also the great weight of the body that has to be sustained, and, at times, the roughness and hardness of the ground over which compulsory locomotion is performed.

Foot lesions affecting cattle give rise to sudden and well-marked lameness in one or more limbs, and disinclination to move or to search for food. If both fore feet are affected the hind feet are frequently drawn up under the body to take the weight, as in laminitis of the horse; and if three or all four feet are affected all are drawn up under the body, the back is arched, and the animal presents a tucked up and distressed appearance. The earliest manifestation of foot lesions is uneasiness and paddling of the feet, indicative of soreness and tenderness. As the lesion matures the animal is constantly picking up the fore feet, snatching, kicking out, and shaking the hind limbs, as if to free some irritating foreign body from the foot; the coronet appears swollen and is tender to manipulation; the superficial vessels

are injected, noticeable in thin skinned and light haired animals ; and the affected foot is hot.

Examination of the foot in cattle, particularly the hind foot, is, at the best of times, attended with a little difficulty and trouble, and, when lesions are present, the animal usually gives increased resistance. It is advisable prior to examination to soak and cleanse the foot with water, which will enable one more easily to detect the presence of vesicles (small), and any recent separation of horn and skin that may have taken place. Small bladder-like elevations can be distinctly seen on the surface of the coronet, close to the junction of the skin and horn, usually towards the heel, varying from the size of a small pea and upwards, or several small vesicles may be seen in a line, which coalesce to form one large fluctuating painful swelling. This ruptures and the vesicular contents escape, exposing a red surface. In the case of foot lesions the red base is not so bright in colour as in lesions affecting the buccal mucous membrane, and the interdigital base of the lesion is still less intense in colour ; in small lesions the base appears as a red spot. There is no tendency to suppuration, unless aggravated by dirt or complicated by other external influence.

Where lesions are large a well-marked separation of the horny and sensitive foot can be detected, extending from above downwards, never *vice-versa*, and the horn always remains normal in appearance.

Vesiculation also occurs in the interdigital space, near to the superior border of the wall, extending in a backward or forward direction ; at times a large, confluent vesicle is formed from the coalescing of several smaller vesicles situated at various points in this space, between the anterior and posterior commissures of the foot. Vesicles are formed immediately adjacent to the anterior commissure, or anywhere adjacent to the coronary band. They may be single or multiple, and, when they rupture, they give an appearance to the part of isolated tufts of matted hair of brownish colour, which, if removed, reveal the characteristic surface of the lesion. Extensive lesions causing complete disunion of horn and skin are often associated with much swelling of the coronet, extending to the fetlock joint and above, and the animal apparently experiences great pain. Small vesicles in individual cases can be seen around the ergot.

In severe and aggravated cases suppuration is present, extending in a downward direction under the wall, and even the sole of the foot is implicated at times ; the leg swells up to a considerable size, and numerous small abscesses may appear between the foot and axilla or groin respectively. The hoof in these cases is commonly cast, and the animal takes many weeks or months to recover, owing to the slow growth of new horn, or it may die in the meanwhile from pyæmia or from exhaustion.

In milch cows attacked by the disease the udder is frequently the seat of lesions. Vesicles form on the udder or any part of the teat as small isolated bladder-like elevations, or as larger, circular or oval, well-defined, fluctuating swellings, extending over a large surface of the teat, and occasionally reaching and giving rise to occlusion of the external orifice of the duct. The undisturbed vesicle bursts, while the exudate (in many cases tinged with blood) collects and coagulates on the surface of the lesion. When this coagulum together with the

thin portion of epithelium is removed the typical red surface is exposed. In large ruptured lesions which involve a greater part or the whole of the teat an intense hyperæmic condition is present, and the base is surrounded by ragged pieces of epithelium still adherent to the teat.

The hyperæmic condition of the surface of the teat is often aggravated by the process of milking, which retards repair and promotes bleeding, giving rise to the formation of large sores or ulcers, or, on entrance of pyogenic organisms, to suppuration of the part.

Calves affected with the disease frequently succumb before any appreciable manifestation of sickness or lesions are present. Death is particularly frequent in calves receiving the warm milk from cows affected in the udder, and, more especially, sucking calves. The symptoms and lesions when present are similar to those affecting adult cattle.

SYMPTOMS AND LESIONS IN SHEEP.

Sheep attacked by the disease suffer more severely than cattle. The evidence of contagion amongst the flock is well marked, large numbers often becoming affected in the course of twenty-four to forty-eight hours. The loss in condition is so rapid that they may be said to "melt like butter before the sun"; it is really astonishing how marked and sudden is this unsightly change, the sheep in many instances looking most pitiable objects.

Foot lesions are most constant, severe, and extensive; it is not often that one foot is affected (as in foot rot), but usually two or more.

The animal trembles and totters along, unable to walk, hardly knows which foot to place to the ground, picks its way, takes the body weight off the affected feet most cautiously, often falling down exhausted in the attempts to guard itself, walks like "a cat on hot bricks," and appears in much pain and greatly distressed.

At pasture sheep are found lying scattered about the ground, some in remote corners, under hedgerows, etc., and refuse to rise.

The temperature is high and the respirations often so rapid that the animal pants, especially during hot weather.

Some sheep go down on their knees to feed, others feed lying down resting on the sternum or dragging themselves about, unable to rise or stand, and falling down in the attempt.

Being unable to search for food, and owing to intense pain of the foot lesions, they speedily present a most emaciated and dejected appearance.

Other affected sheep that are standing are very restless, moving from one foot to the other, constantly picking up the fore feet and snatching the hind foot close up to the abdomen, kicking out of the hind limb. One hind leg may be held up for a long time, lessening the tension of the flexor muscles, but the animal is careful not to bring the affected foot in contact with the ground. When compelled to, it rests on the toe or anterior wall of the foot, and the back is arched. The position and guarded movements of the sheep truly indicate the intensity of the affection to which it has become a victim. Owing to the smallness of the foot lesions usually implicate the greater part of it, and partial separation of the horn and skin is well

marked in most cases. The heel is the chief seat for formation of vesicles; these appear as one or several small vesicles, which often coalesce.

Small vesicles may not be larger than a large pin's head, appearing when burst as small punctures in the skin of the part, but close examination reveals their true nature. In sheep that appear lame, but do not show any apparent vesiculation, if the heels are taken between the fingers and thumb and pressure is applied a spurt of a few drops of fluid may often be observed, and when the pressure is relaxed from the part small ruptured lesions can be seen as well-defined punctures in the skin. The lesions appear at any point adjacent to the coronary band, often on the anterior part of one or more digits, and in the interdigital space. In a typical case there is no disease of horn tissue, although foot-rot may be associated with this disease in the same animal, and then the foot-rot is responsible for the diseased horn.

There is no suppuration or granulation of tissue (fungoid growth) unless the parts become aggravated by dirt, when the hoof is frequently cast (always in its entirety), exposing the intensely hyperæmic sensitive laminæ, which readily bleed.

The course of the foot lesions and the local changes of the part are similar to those described as affecting cattle.

Mouth lesions are not nearly so common in sheep, and when present are usually not so extensive as in cattle.

Lesions in the mouth may, however, appear under the mucous membrane of the lips, tongue, pad, hard palate, or cheeks, usually very small in size, and indicated after having burst by red spots, which rapidly heal, causing little or no inconvenience to the patient. The best marked lesions affect the pad, giving to it a raised appearance. The raised and separated mucous membrane peels off as one scaly crust, exposing a slight pink-tinted surface, which soon loses the delicate colour, the affected part in a very short time appearing quite normal. The exudate is always small in amount. Any appreciable salivation or "smacking of the lips" is absent, but there is a frequent snapping motion of the lips and grinding of the teeth; a foetid condition of the breath and diarrhœa are often present.

Small vesicles on the mammary glands and teats similar to those seen in cows may be detected, but are not so common.

SYMPTOMS AND LESIONS IN PIGS.

Pigs exhibit rise in temperature and general constitutional disturbance. The animal shows disinclination to move, hides itself under the litter, when disturbed cries out as if in great pain, and if compelled to walk goes most cautiously on the toes. The feet are hot and tender to manipulation, and the legs are swollen to above the fetlock joints. Vesicles, as a rule, appear around the coronary band or interdigital space, close to the pads of the feet, and they sometimes involve the entire foot, or all feet.

Lesions affecting the mouth of pigs are more often confined to the snout than the mucous membrane of the mouth, but occasionally vesicles of large size are found on the tongue and pharynx, the latter lesions being responsible for suffocation and death of the animal.



Lesions on the abdomen sometimes involve the entire surface of the mammary glands, in the form of numerous small vesicles.

Sucking pigs, like calves, die suddenly from drinking the contaminated milk of the cow, without showing any symptoms of disease.

Although almost all cases of foot-and-mouth disease exhibit symptoms and lesions of an unmistakable nature, it occasionally happens that individual animals do not fully present the characteristic and typical combination of symptoms and lesion that we would desire.

A careful enquiry and examination may be necessary to distinguish from other affections to similar parts arising from such causes as injuries from the presence of foreign bodies, coarse food stuffs, irregular teeth, granulations of the parts, actinomycosis, pustular and ulcerative eruptions of the mouth, foot-rot, foul in the foot, wounds and lacerations, and the action of corrosive agents—medicinally or otherwise employed.

Finally, by taking into consideration the history of the case, the absence or presence of several seats affected, the evidence, if any, of contagion amongst the stock, and by the valuable aid of the thermometer extended to other than apparently affected animals, it is generally possible to arrive at an accurate diagnosis.

DESCRIPTION OF PLATE II.

FIGS. 1, 2, and 3.—Portions of membrane covering the hard palate, taken from three individual cattle, showing well-defined ruptured vesicles, for the most part circular in shape. In Fig. 2 the vesicles are multiple, with a tendency to coalesce.

FIG. 4.—The upper lip and dental pad of a bullock, showing the seats of ruptured vesicles near the junction of the upper lip and dental pad.

FIGS. 5 and 6.—Extensive lesions resulting from vesiculation, showing the free portion of the tongue completely denuded of its mucous membrane. In Fig. 6 the mucous membrane is hanging from the tip, and the presence of papillæ can be distinctly seen on the denuded surface of the tongue.

FIG. 7.—Tongue of bullock showing three seats of ruptured vesicles, varying a little as regards the stage of the lesions, viz.: (a) recent and irregular in shape; (b) of earlier date, small, circular, and partially cicatrised, a smooth thin layer of epithelium having formed (seen below the level of the surrounding surface); (c) of usual size, circular in shape, and repair not so far advanced as in middle lesion.

FIG. 8.—Portion of bullock's tongue, from about the middle, showing a somewhat extensive ruptured lesion with large base and an irregular edge of ragged epithelium.

FIG. 9.—Lower third of tongue, showing an oval and other lesions in which resolution is rapidly taking place.

SOME IMPORTANT MODIFICATIONS IN THE PREPARATION, PROPERTIES, AND DOSES OF DRUGS ACCORDING TO THE BRITISH PHARMACOPŒIA, 1898.

By H. A. WOODRUFF, M.R.C.V.S., Royal Veterinary College,
London.

THE publication of a new edition of the *Pharmacopœia* is an event of importance to all practising veterinary medicine, involving as it does differences in the methods of preparation of many drugs and their derivatives, eliminating useless and obsolete remedies, and recognising new and valuable agents. In the first place, differences of preparation require in many cases a readjustment of doses; then changes of nomenclature demand attention in prescription writing; and the inclusion of new remedies, and the omission of obsolete ones, afford some guide to the trend of scientific medical opinion, not by any means to be blindly followed in all cases, but always to be wisely considered.

In this article a few points of difference between the edition of 1885 and that of 1898, which seem of most importance to the veterinary therapeutist, are mentioned. One of the most radical changes is that involved in arranging for uniformity of the dose of tinctures, so that, with the single exception of tincture of iodine, all the rest may be divided into two groups, the more potent, such as aconite, belladonna, opium, digitalis, cannabis, indica, nux vomica, of which the dose is ʒij - ʒiv for the horse, and Mv - xv for the dog; and the less potent, such as gentian, calumba, etc., of which the dose remains ʒss - ʒifs for the horse, and ʒfs - ʒj for the dog. Another important change with regard to tincture is that an endeavour has been made to find the menstruum best adapted for extracting the active principles from the drug, and to this end, instead of using rectified spirits in most cases, alcohol (ethyl hydroxide) of five definite strengths, namely, 90 per cent., 70 per cent., 60 per cent., 45 per cent., and 20 per cent., has been used.

In the case of the tinctures of nux vomica, cinchona, and opium, they are required to be of a constant alkaloidal strength, containing, in the first, 1 grain of strychnine in 1 fluid ounce, in the second, 33 grains of opium, or ʒi of morphine per ounce, and in the third, 1 per cent. of total alkaloids (1 grain in 110 minims).

To provide for these changes, a few tinctures have been reduced, and many increased in strength. With regard to infusions, here also the changes have been towards providing greater uniformity, and more infusions have been made with 1 of the drug to 20 of water. A new class of preparations has also been introduced, in the form of concentrated infusions, known as "*Liquores concentrati*." These can be diluted with water if necessary, but will be found convenient in the officinal solution, and the chief of them are calumba and rhubarb, each with 10 ounces of the drug to the pint, and quassia, with 2 ounces to the pint. In all three the dose is ʒi - ʒij for the horse, and ʒss - ʒj for the dog.

Among other additions are the liquid extracts of belladonna, and of nux vomica, in the former of which there is a definite amount ($\frac{3}{4}$ grain in 110 minims) of total alkaloids, and in the latter a definite amount of strychnine ($1\frac{1}{2}$ grains in 110 minims). In the case of most of the other liquid extracts, 1 fluid ounce represents 1 ounce of the drug employed. Other interesting additions are:—liquor ethyl nitritis, an alcoholic solution containing 3 per cent. of ethyl nitrite, and forming a more reliable preparation than spirits of nitrous ether; also cocaine ointment, containing 1 of cocaine in 24 of a mixture of oleic acid and lard; and, for the first time, the compound syrup of phosphate of iron, quinine, and strychnine, familiar under the name of Easton's Syrup.

Among the preparations deleted are the liquor ammonia acetatis fortior, liquor antimonii chloridi, and liquor iodi, all of which are both popular and useful in veterinary practice, and will probably be retained, although now not official.

The changes of nomenclature are unimportant, being chiefly in the direction of simplicity and chemical accuracy. The most noticeable is the use of *hydrochloride* instead of *hydrochlorate*, to express the salt of an alkaloid such as cocaine hydrochloride.

Of more importance are the alterations in composition and in strength. Examples of the former are the *injectio morphinæ*, in which the tartrate of the alkaloid replaces the acetate, and which is made only half the strength of the old preparation—*i.e.*, 5 grains in 110 minims, instead of 10 grains in 100 minims; and *tinctura chloroformi et morphinæ*, commonly known as *chlorodyne*, in which sulphuric ether has been omitted, and the quantity of morphine multiplied by four and a half. The preparation now contains half a grain of morphine in each fluid drachm, and the menstruum is 90 per cent. alcohol instead of syrup.

The alterations in strength, requiring adjustment of doses, are, besides the tinctures, infusions, and preparations alluded to above, *aqua chloroformi*, which is reduced to half the old strength—namely, to 1 in 400 of water; and *injectio apomorphinæ*, which is now made to contain 1 grain of the hydrochloride in 110 minims of distilled water, instead of 2 grains in 100 minims.

In view of some of the above important changes in dosage, particularly in the tinctures, no apology is needed for emphasising the necessity of carefully labelling all stock medicines with the date of the Pharmacopœia to avoid confusion otherwise inevitable.

EXPERIMENTS REGARDING THE IMMUNISATION OF CATTLE AGAINST TUBERCULOSIS.

By J. M'FADYEAN, Royal Veterinary College, London.

WHEN the common domesticated species are arranged in the order of their susceptibility to tuberculosis the first place is by general consent assigned to bovine animals. Their claim to that position appears to be incontestable in view of the large proportion of them that contract the disease in what may be called natural, as opposed to experimental, conditions. This may be admitted in spite of the fact that the proportion of victims furnished by the different species is only a rough measure of their susceptibility, owing to the varying conditions under which they are kept. The sheep is probably less susceptible than the ox, but the difference between these two species in this respect is perhaps not so great as the rarity of the disease in the one and its frequency in the other would at first sight appear to indicate. If it were desired to measure the difference of susceptibility with some approach to accuracy it would be necessary to resort to experiments on a considerable scale.

Do all individuals of the common breeds of European cattle share in the susceptibility which appears to be an attribute of the species regarded as a whole, or do any of them possess such natural powers of resistance to infection as would entitle them to be called immune? Speaking broadly, it may be said that when considerable numbers of animals are dealt with the number that fall victims to the disease is proportional to the extent to which the conditions of their environment have been favourable to infection, and to the length of their exposure to such conditions. In exceptional cases all the individuals in a herd of considerable size contract the disease, but the rule is that some escape. Do these fortunate individuals owe their escape to powers of resistance above the average—in other words, to a practically useful degree of immunity, or to the fact that in spite of the apparent uniformity of the conditions they have had to offer resistance to a smaller quantity of infective material than the animals of the same herd that contracted the disease? It may safely be said that experimentation is necessary to solve this question.

Another interesting question that presents itself in this connection is: Do individuals of the bovine species acquire immunity as the result of recovery from a first attack of tuberculosis, whether contracted naturally or induced experimentally? Probably a negative reply to this question would be in harmony with the views most generally accepted hitherto, but caution would suggest a guarded answer until further evidence is available. It is true that tuberculosis has frequently been cited as an example of a disease against which no immunity is conferred by a first attack, but the only evidence in support of the conclusion is drawn from clinical observation on human beings who are alleged to have had successive attacks of the disease. In such cases, however, it is in the highest degree probable that the interval during which the patient was supposed to have made a complete

recovery was merely one of partial retrogression of the lesions. At any rate, the only evidence that would be at all convincing in such a case would be that furnished by the application of the tuberculin test to the supposed convalescent, or by a careful *post-mortem* examination of all his organs. During recent years the application of the tuberculin test to animals of the bovine species has in some instances appeared to point to the complete recovery of some individuals, but, so far as the writer is aware, no reliable evidence of the re-infection of such animals has been placed on record, nor has evidence of increased resistance to infection after apparent recovery been sought for experimentally.

In the following pages there will be described a number of cases in which animals of the bovine species were found to possess a very high degree of immunity to tuberculosis, but, as will presently appear, the circumstances make it difficult or impossible to determine whether the immunity was natural or acquired, and, if acquired, whether it was the result of recovery from the first attack or attributable to the tuberculin with which the animals had been tested or treated.

CASE I.—The subject in this case was one of the animals employed in the experiments carried out for the Committee appointed by the Royal Agricultural Society with regard to the reliability of the tuberculin test.¹ It was a yearling shorthorn heifer, purchased on the 30th September 1899. It was first tested with tuberculin on the 9th October, and it reacted distinctly, the temperature rising to 106° at the twelfth hour. The test was repeated on the 12th of the same month, and it again reacted, the maximum temperature recorded being 106·2° (at the twelfth hour). Subsequent tests with the same quantity of tuberculin (1 cc.) were made as shown below:—

<i>Date.</i>	<i>Max. Temp.</i>
Oct. 24	103·4
" 26	105·6
Nov. 1	102·2
" 10	104·5
" 21	103·3
" 28	102·6
Dec. 9	102

It was decided to use larger doses of tuberculin with a curative object, and the particulars of these injections were as follows:—

<i>Date.</i>	<i>Dose.</i>	<i>Max. Temp.</i>
Dec. 22, 1899	10 cc.	100·6
" 26 "	"	102
" 28 "	"	102·6
Jan. 1, 1900	"	102·1
" 10 "	20 cc.	102·3
" 13 "	"	102
" 17 "	"	102
" 21 "	"	102
" 24 "	"	101

¹ It forms No. 13 in the Committee's Report. See Vol. XIII. of this Journal, p. 373.

On the 6th February 2 cc. of a liquid that was very rich in tubercle bacilli was injected into one of the jugular veins of the animal. The liquid was prepared by rubbing up some caseo-purulent matter from the mesenteric gland of a horse with sterile water. On the same day two other animals of about the same size and age were infected in the same way with the same quantity of the same material. It is unnecessary to refer further to these two animals, except to say that they subsequently became ill, and when they were killed on the 10th April their lungs were found to contain numerous tubercles, while the bronchial and mediastinal lymphatic glands were greatly enlarged and caseating.

On the 14th and 17th February the animal, which had previously failed to react even with 20 cc. of tuberculin, reacted distinctly to the test with 1 cc. Between the 24th February and the 28th March it was treated with doses varying from 5 cc. to 20 cc., and on none of these occasions was there any important rise of temperature after the injection. After the last-mentioned date it was left untreated, and on the 22nd May following it was killed. A most searching *post-mortem* examination revealed no lesion anywhere except in one mesenteric gland, which contained a completely calcified tubercle about the size of a pea. It appears to be fair to assume that this lesion had been in an active condition when the animal was purchased, and that it was accountable for the reactions at first obtained.

It is impossible to doubt that this animal had latterly a very high degree of immunity against tuberculosis, since the intravenous injection of a large quantity of virulent tubercle bacilli caused it no apparent inconvenience, and entirely failed to set up any permanent lesion in the lungs or elsewhere. Unfortunately, the circumstances were too complex to enable one to draw a very positive conclusion as to the way in which the immunity came about. It is conceivable, but very improbable, that the animal had a high degree of natural immunity; the fact that it had contracted the disease naturally is against this view. It appears to be much more likely that the immunity was acquired while the animal was under experiment, but it is not possible to say with any confidence whether the credit of this should be ascribed to an immunising effect of the tuberculous lesion in the mesenteric gland, or to the injections of tuberculin. Possibly both of these may have been factors in the production of the immunity.

CASE II.—This animal has had a history very similar to that just described. It is an Ayrshire heifer, which was about a year and a half old when it was purchased on the 30th September 1899. It was tested with tuberculin on the 9th and 12th October, and it reacted distinctly on both occasions. On the 27th November following it was again tested, and the temperature rose from 101.1° to 103° (ninth hour). On the 8th January 1900 it was tested for a fourth time, and the maximum temperature during the following fifteen hours was 106° . On each of these occasions the dose of tuberculin injected was 1 cc.

On the 11th January $1\frac{1}{2}$ cc. of a liquid rich in tubercle bacilli was injected into one of the animal's jugular veins. The liquid was prepared by rubbing up a portion of the tuberculous liver of a rabbit with sterile bouillon, and allowing the coarser particles to settle.

The rabbit had been inoculated intravenously on the 6th December with tubercle bacilli from a caseous bronchial gland of an ox, and its liver was crammed with miliary tubercles. On the same date (11th January) another heifer of about the same size and age was made the subject of a parallel experiment. This control animal was killed on the 2nd March following, when it was very ill, and the *post-mortem* examination revealed a dense miliary tuberculosis of the lungs.

The other heifer was treated with tuberculin in 20 cc. doses on the 15th, 17th, 21st, and 24th January, 28th February, and 8th March, and on none of these occasions was there any rise of temperature amounting to a reaction. On the 15th March it received 10 cc., and on the 28th March 20 cc., again without any distinct reaction.

On the 5th April 1900 the heifer was again inoculated intravenously with tubercle bacilli. The material for inoculation was in this instance prepared by triturating a portion of the fresh lung of a rabbit dead from tuberculosis (as the result of an intravenous inoculation) with sterile water, and allowing the coarser particles to settle. A cover-glass preparation made from the liquid showed numerous tubercle bacilli, and the quantity of the liquid used for the inoculation was 2 cc. The material employed for the inoculation of the before-mentioned rabbit was taken from the tuberculous pharyngeal gland of an ox.

The intravenous injection did not occasion any recognisable disturbance either at the time or afterwards, but the heifer reacted distinctly to an ordinary dose of tuberculin when tested on the 13th June, 18th July, and 16th October following. On the last of these occasions the temperature rose from 101° to 105·4° (at the fifteenth hour). The test was repeated on the 20th November, when there was an indecisive rise of temperature.

On the 28th November 1900 a third intravenous injection of tubercle bacilli was administered to the heifer. The liquid for inoculation was prepared by triturating a quantity of caseo-purulent matter from the tuberculous spleen of a horse with sterile water, and allowing the coarser particles to settle. The quantity of the supernatant liquid injected into the heifer was 2 cc., and a cover-glass preparation made from it showed that it was very rich in tubercle bacilli. As a control experiment a rabbit was inoculated intravenously with two drops of the same liquid, and it died on the 12th April 1901, with numerous tubercles in its lungs and kidneys. The heifer was tested with tuberculin on the 19th December following, with the result shown below:—

<i>Time of injection.</i>	<i>6th hour.</i>	<i>9th hour.</i>	<i>12th hour.</i>
102	105	104·2	102·6

An almost identical reaction was obtained when the test was repeated on the 22nd January.

On the 30th January last the heifer was again inoculated intravenously, 5 cc., of a rich emulsion of tubercle bacilli (from an artificial culture) in sterile bouillon being injected into the right jugular vein. As a control experiment the emulsion was further diluted with five volumes of sterile bouillon, and $\frac{1}{2}$ cc. of the diluted liquid was injected into the auricular vein of a rabbit. This rabbit died on the

1st March, and the *post-mortem* examination revealed a dense crop of miliary tubercles in the lungs, liver, and spleen, and a few tubercles in the kidneys.

On the day following the intravenous injection the heifer's temperature had risen to 104.2° , and it remained at about that level till the 4th February, when it had fallen to 99.6° .

During the following three months the heifer appeared to be in good health, and its general condition remained good. On the 15th May it was tested with an ordinary dose of tuberculin, and the maximum temperature recorded during the following fifteen hours was 102.8° (original 102°).

On the 18th May the heifer was again inoculated intravenously with 5 cc. of a rich emulsion of an artificial culture of virulent tubercle bacilli. On the previous day six guinea-pigs had been inoculated intraperitoneally with $\frac{1}{2}$ cc. of a considerably thinner emulsion from the same flask of artificial culture. One of these died from tuberculosis in three weeks, and when the others were killed on the 14th June they were all found to be extensively tuberculous.

On the evening of the 18th May the heifer's temperature was found to have risen from the normal (101° on the 17th) to 105.6° , but it gradually fell, and had returned to the normal on the 21st May. Between the latter date and the 18th June it never reached 102° . On the 18th June the animal was tested with an ordinary dose of tuberculin, with the result shown below :—

<i>Time of injection.</i>	<i>6th hour.</i>	<i>9th hour.</i>	<i>12th hour.</i>
101.6	103	102	101.4

At the present time the heifer appears to be in perfect health, and its general condition is good.

CASE III.—The subject of this experiment is a Hereford heifer, now three years old. It was first tested with tuberculin on the 5th May 1899, and there was then an entire absence of reaction.

On the 10th May 1899 the animal, then about a year old, was inoculated intravenously with $2\frac{1}{2}$ cc. of a liquid rich in tubercle bacilli. The bacilli were obtained from the tuberculous liver of a pheasant, which had contracted the disease naturally. A cover-glass preparation made with a trace of the liquid used for inoculation showed tubercle bacilli in every field of the microscope. The animal was tested with tuberculin on the 11th and 20th May, and on these occasions there was no reaction. The test was repeated on the 31st May, with the following result :—

<i>Time of injection.</i>	<i>6th hour.</i>	<i>9th hour.</i>	<i>12th hour.</i>	<i>15th hour.</i>
102.8	103.8	104.4	105	105

There was thus a distinct reaction to the tuberculin test on the twenty-first day after infection. It was then resolved to repeat the test at short intervals. The following table shows the results of successive tests :—

[TABLE.

<i>Date.</i>	<i>Time of injection.</i>	<i>6th hour.</i>	<i>9th hour.</i>	<i>12th hour.</i>	<i>15th hour.</i>
June 13	101	102'8	105	105'6	105
" 22	102'8	103'4	104	104'8	104
" 29	101'4	103	104'2	105	105
July 7	102'4	103	104	104'8	104'4
" 15	101'4	103'2	104	104	103'8
" 22	102	104	102'8	102'5	102'4
" 28	101	104	103'8	102'4	101'6
Aug. 4	101'4	103'6	103'8	104	103'2
" 11	101'9	102'7	104'6	101'4	100'8
" 18	103	103'3	103'3	102'9	103
" 25	102	102'9	103'4	103'1	101'6
Sept. 1	101'5	103	103	102'5	101'3
" 8	103'2	103'8	103'6	102'2	102
" 16	102	103'5	103'6	103	102'2
" 24	101	103'4	102'6	101'8	101'2
Oct. 26	102	101'4	102	101'9	101'8

On the 11th November the heifer was again inoculated intravenously, this time with $2\frac{1}{2}$ cc. of liquid containing tubercle bacilli from the liver of a rabbit. The rabbit had died twenty-six days after intravenous inoculation with material from the tuberculous mesenteric gland of a horse, and its liver was crammed with miliary tubercles which were very rich in tubercle bacilli.

The heifer was tested with an ordinary dose of tuberculin on the 21st and 28th November, and there was no reaction. The test was repeated on the dates and with the results shown below :—

<i>Date.</i>	<i>Time of Injection.</i>	<i>6th hour.</i>	<i>9th hour.</i>	<i>12th hour.</i>	<i>15th hour.</i>
Nov. 21	103	102	101	101	100'4
" 28	102	103'4	101	102'4	102'4
Dec. 9	101'3	102	101'4	101	100'6
" 22	101'7	101'7	101'9	102	101'2
Jan. 8	101	102'6	102	101'5	102

On the 11th January 1900 the heifer was inoculated intravenously for the third time, $1\frac{1}{2}$ cc. of a liquid containing tubercle bacilli (same as used on that date for the subject of Case II.) being injected into one of the animal's jugular veins. As in the previous cases, the injection did not occasion any obvious disturbance, and the animal remained in apparent good health.

On the 5th April a fourth intravenous injection of tubercle bacilli was given. The liquid injected was the same as that used on the same date for the subject of Case II., but the quantity injected was 3 cc. No symptoms of illness followed the operation, but when the animal was tested with tuberculin on the 13th June, 18th July, and 16th October following it reacted distinctly.

The 20th November was the date of the next test, and on that occasion there was no reaction, as shown below :—

<i>Time of Injection.</i>	<i>6th hour.</i>	<i>9th hour.</i>	<i>12th hour.</i>	<i>15th hour.</i>
101'2	101'5	101'8	101'8	101'4

On the 28th November 1900 the heifer was inoculated intravenously for the fifth time. The material employed was the same as that used on the same date for the subject of Case II., and the quantity of liquid injected was the same, viz., 2 cc. On the 29th November, the animal's temperature was $101^{\circ}6'$, and during the next three weeks it never exceeded 102° . On the 7th December, the heifer was tested with tuberculin, and the result was as follows:—

<i>Time of Injection.</i>	<i>9th hour.</i>	<i>12th hour.</i>	<i>15th hour.</i>
101'4	101'8	102'6	102

The test was repeated on the 19th December and 22nd January and there was an entire absence of reaction.

On the 30th January of this year a sixth intravenous injection of tubercle bacilli was administered, the material and the dose being the same as was used in the previous case on that date. On the same evening the temperature had arisen from 102° to $104^{\circ}2'$, and it remained over 104° during the next four days. On the 5th February it had fallen to 102° , and, subsequently, it remained about the normal. The animal was tested with tuberculin on the 12th February, and the result was as follows:—

<i>Time of Injection.</i>	<i>9th hour.</i>	<i>12th hour.</i>	<i>15th hour.</i>
101	102'4	101'2	101

On the 23rd March last the heifer was inoculated intravenously for the seventh time. In this instance the material for injection was prepared by taking a quantity of the surface growth from a bouillon culture of the tubercle bacillus and rubbing it up with sterile water. The liquid was decanted into a test tube, and the coarser clumps of bacilli were allowed to deposit. The supernatant liquid was still distinctly hazy from suspended bacilli, and the quantity of it injected into the heifer's jugular vein was 15 cc. As a control experiment a rabbit was inoculated intravenously with two drops of the same liquid. The rabbit was killed on the 25th April, and its lungs were then found to contain numerous pin-head tubercles, in which tubercle bacilli were abundantly present.

On the evening of the 23rd March, the heifer's temperature had risen from the normal to $105^{\circ}4'$. On the 24th, it was 103° , and on the following day it had fallen to about the normal (102°). The animal continued in apparent health, and when it was tested with tuberculin on the 15th May the maximum temperature was $102^{\circ}8'$ (sixth hour).

On the 18th May last, the heifer received an eighth intravenous injection of tubercle bacilli. The material and dose were the same as in the preceding case on the same date. On the evening of the 18th May the temperature had risen to $105^{\circ}6'$, but on the 20th it had returned to the normal. On the 17th June it was 102° , and on the following day the animal was tested with tuberculin; there was no distinct reaction, the maximum temperature recorded during the following fifteen hours being 103° (sixth hour). At the present time the animal appears to be in good health, and its general condition is fair.

CASE IV. It may suffice if the particulars relating to this case are given in a less detailed form. The animal is a shorthorn cow, now

about six years old. It was tested with tuberculin on the 19th September 1898, and there was no reaction. On the 29th of the same month it was inoculated intravenously with 4 cc. of sterile water holding in suspension tubercle bacilli from the liver of a fowl (natural case of the disease). Microscopic examination showed that the liquid was rich in tubercle bacilli.

The cow was tested with tuberculin on the 7th October following, and the result was as shown below :—

<i>Time of Injection.</i>	<i>8th hour.</i>	<i>10th hour.</i>	<i>12th hour.</i>	<i>14th hour.</i>
101·8	105	105·4	105·4	105

There was an equally pronounced reaction when the test was repeated on the 17th October and 18th November, but the animal failed to react when tested on the 15th December. Since that date it has been re-inoculated intravenously with tubercle bacilli as follows :—

11th November 1899, with 4 cc. of the liquid used on that date for Case III.

5th April 1900, with 3 cc. of the same liquid as was used on that date for Cases II and III.

23rd March 1901, with 15 cc. of the liquid used on the same date for Case III.

18th May 1901, with 5 cc. of the same liquid as was used on that date for Cases II. and III.

At the present time this animal is very fat ; it appears to be in perfect health, and it does not react to tuberculin.

ORIENTAL OR BUBONIC PLAGUE IN CERTAIN OF THE LOWER ANIMALS.

By WILLIAM ROBERTSON, M.R.C.V.S., Government Veterinary
Surgeon, Agricultural Department, Cape Town.

ALTHOUGH a great amount has been written during the past few years upon the history, clinical or otherwise, and bacteriological and microscopical examination of bubonic or oriental plague amongst the human race, with the exception of the rat, I can find little or no mention of the fact that other of the lower animals share with man a natural susceptibility to the disease. I purpose describing certain cases of bubonic plague which have been brought to my notice in the cat, rat, ferret, guinea-pig, and mouse, during the epidemic of the disease in Cape Town.

CATS—A cat was brought to the laboratory by a man who said he had seen it sitting huddled up in a garden at the back of a house in which had been found rats dead from plague, and he wished to know if the cat was suffering from the same disease. The animal was a wretchedly poor mongrel, very thin, and unable to stand ; head, particularly under the lower jaw, very much swollen, and eyes closed, thus giving the whole head a most peculiar triangular appearance.

On palpation, I at first suspected an abscess, but owing to the diffuseness of the swelling I dismissed this idea. After destruction of the animal (with chloroform) I made a *post-mortem* examination, and found both submaxillary lymphatic glands to be enormously enlarged and swollen, a pale straw-coloured oedema surrounding them, and involving the tissue in the submaxillary space as far down as the trachea. On cutting into their substance, the glands were seen to be spongy, hæmorrhagic in patches, with no signs of supuration, and on squeezing a white fluid exuded. Smears from the glands, stained by Loeffler's methylene-blue, or weak carbol-fuchsin, showed the characteristic bipolar-staining bacilli of oriental or bubonic plague, and these bacilli were much more characteristic than those generally found in the bubos in human cases of the same disease. The spleen was enlarged and deeply congested, while the liver was congested, and smears from both these organs contained characteristic bacilli.

I have made *post-mortem* examinations on several cats dead and dying from plague, and in all the main lesion seemed to be enlargement of the submaxillary glands, due, no doubt, to a primary infection as a result of eating rats dead or dying from plague. In several of the cases I found the presternal lymphatic glands enlarged and teeming with typical bipolar-staining bacilli.

RATS.—It has often happened that many days or even weeks previous to the outbreak of plague in a neighbourhood rats have been noticed to be sick, and people will tell you these animals are then not so quick in their movements, and are more easily knocked over with a stick. Rats dead from plague generally show distinct enlargement in the glands of the neck below the jaw, less frequently in the presternal group or in the groin. On *post-mortem* examination the swelling of the neck is found to be due to an extensive oedema of the subcutaneous cellular tissue, the thyroid and submaxillary glands are about twice their normal size, and in their neighbourhood the lymphatic glands are enlarged to about the size of a pea or bean, deeply congested, with hæmorrhages into and around them.

The presternal lymphatic glands (if the rat has been able to hold out long enough) seem to be the next group affected. Bacilli possessing typical bipolar-staining properties, and which do not stain by Gram's method, are found in immense numbers in the hæmorrhagic lymphatic glands.

In every case I have also found the spleen enlarged, the liver congested, and bacilli present in smears from both these organs.

FERRETS.—Two ferrets which had been used to clean out rats from a plague infected area were noticed to be sick, refusing food, coat staring, inclined to sleep, and with a muco-purulent discharge from the eyes. In from eight to thirteen days as far as could be made out after exposure to infection these animals died.

On *post-mortem* examination a complete chain of swollen, congested, and in places hæmorrhagic, glands, embedded in a clear yellow gelatinous oedema, could be made out, extending on each side of the trachea, and in one case a presternal gland was enlarged and approaching supuration. Cover glass smears made from these glands contained bacilli, indistinguishable from the plague bacilli.

In both animals the spleen was about twice the normal size, deep

purple in colour, and contained the same bacilli, not, however, quite so numerous as in the glands.

In one of the ferrets I found an unhealthy-looking ulcer in the neck, probably as the result of a bite from a rat.

It may be of interest to note that these ferrets were housed near some guinea-pigs, three of which died suddenly, presenting similar *post-mortem* lesions—swollen and hæmorrhagic glands surrounded by œdema.

In these guinea-pigs I found similar organisms showing bipolar-staining, and producing stalactite growths in bouillon, and I reproduced a similar disease in other guinea-pigs by inoculation with a culture in bouillon.

MICE.—Comparatively few of these animals have been brought to me for examination, but in two out of the number I have found enlarged submaxillary and presternal glands deeply congested, and containing bacilli resembling *B. Pestis*. I was surprised to find that the blood in both cases contained an enormous quantity of the same organism. Unfortunately I did not have an opportunity to make cultures from these animals.

I should like while finishing these few notes to add that I have had brought to my notice lately an unusual number of outbreaks of disease amongst poultry in the Cape Town district, and in all the specimens submitted I have found the bipolar-staining bacillus of chicken cholera. Many of these bacilli so closely resemble the *B. Pestis* as to deceive experts in human plague. But in culture this fowl cholera organism does not produce stalactite growths. I have been informed that in Bombay during the plague epidemic pigeons died from the disease. It is a matter of conjecture whether there is any connection between the disease in poultry and the disease oriental plague in man.

EDITORIAL ARTICLES.

INFLUENZA OR PINK-EYE?

IT is impossible to escape a feeling of curiosity as to the origin of the movement which brought together the deputation that recently waited on the Minister of Agriculture in connection with the subject of influenza and pink-eye. It would be very natural to suppose that the eminent representatives of the horse-breeding interest who formed the deputation must have in large measure relied on some member or members of the veterinary profession for the information which they desired to lay before the Minister of Agriculture; but, on the other hand, some of the statements that were made strongly suggest that it was purely a layman's deputation, and that its members were very imperfectly informed with regard to what they would probably have called the "facts of the case."

From the reports which appeared in the daily papers, we gather that the burden of the complaint which the deputation desired to lay before the President of the Board of Agriculture was that the breeders and owners of horses are suffering serious losses that are ascribable to the importation of foreign horses suffering from "influenza or pink-eye." It was represented that many of the horses imported from America were affected with one or other of these diseases at the time of landing, and that they carried the contagion and "spread ruin and disaster amongst valuable home-bred, thorough-bred and carthorse stock." Strange to say, the speakers were not in a position to cite any concrete case in which this had occurred, but one of them testified that "he had himself seen these American horses in a large repository in England reeking with influenza." The same speaker anticipated the objection that the diseases in question were always more or less prevalent among home-bred horses, and endeavoured to dispose of it by observing that the same argument might have been employed to stay the hand of the Board of Agriculture in dealing with diseases of cattle, sheep, and pigs with which the Board had successfully grappled.

It is worthy of note that all the speakers used the expression "influenza *and* pink-eye," thus implying that these terms cover two distinct diseases, and one of them took the trouble to explain to the President of the Board of Agriculture that this was so, pointing out that influenza was a very old disease in this country, but that pink-eye was a comparatively modern disease imported from America. This, of course, is a mere matter of terminology, and no doubt at

the present time a good many veterinary surgeons employ the words "influenza" and "pink eye" in the same sense as the members of the deputation. We think, however, that it would be better to regard the two words as synonymous, and to employ the term "contagious equine pneumonia" to the disease which the members of the deputation probably had in mind when they spoke about "influenza." At any rate, it is perfectly clear that before engaging in any dispute as to the advisability of enforcing certain prophylactic measures it would be well to make sure that all those who take part in the controversy have the same diseases in mind and are calling them by the same names.

Now what are the facts with regard to the etiology of these two diseases that ought to be kept in mind in attempting to devise measures for their eradication, which was the ostensible object of the deputation to the Minister of Agriculture? The first of them is that, whatever may have been the source from which they were first introduced into Great Britain, they are now constantly with us, and have no more title to be regarded as exotic than glanders or strangles. No doubt cases of both diseases are occasionally imported, just as cases of glanders are, but when the causation of either is broadly regarded it may safely be affirmed that the entire prohibition of the importation of horses from abroad would not sensibly affect the total number of cases occurring annually. There is not a city of any size in the kingdom in which contagious equine pneumonia has not already found a footing, nor is there a repository into which a horse can be introduced without risk of its contracting the disease. One can readily believe that American horses have been seen standing in an English stable that was "reeking with influenza," but it would be a very hasty inference to conclude that the American horses had introduced the disease. There would, at any rate, be no difficulty in finding cases in stables in which no foreign horse has ever set a foot.

On the part of a deputation that desired only to advocate the imposing of restrictions on imported horses, it showed more courage than discretion to recall the fact that the diseases in question are not uncommon at home, and it was a singularly inept argument to say that in the case of other diseases the Board of Agriculture had not allowed this consideration to deter them from dealing with the question of foreign importation. As every one knows, the demand for the exclusion of foreign cattle, sheep, and pigs found its strongest, and indeed its only, justification in the fact that the country had already made, and was continuing to make, great sacrifices with the object of stamping out certain diseases of these species at home. The position of the deputation would have been defensible and logical had they at the same time urged the introduction of measures calculated to check the spread of contagion at home, and the fact that

they did not ask that any action should be taken in this direction can only be explained by supposing that they had been entirely misinformed with regard to the principal cause of the prevalence of the two diseases in question in this country. At the same time it is very likely that if the various societies that moved in the matter had been better informed on this point the President of the Board of Agriculture would have been left undisturbed, for, while there is no mystery as to the way in which the two diseases are spread, there are immense difficulties in the way of imposing efficient measures of prevention. It would perhaps be going too far to say that the stamping out of influenza and contagious equine pneumonia is impossible, but it may safely be said that the measures necessary to that end would not be tolerated by horse-owners and horse-breeders. The slaughter of all diseased and suspected horses might not be necessary, but compulsory notification, prolonged isolation, and restrictions on the movement of horses in infected areas would certainly be required.

But although the complete eradication of the contagious diseases of the horse, other than glanders, is a problem which the century just begun is likely to hand on to its successor, it does not follow that it is wise, as at present, to allow complete freedom to the agencies by which the specific contagia are mainly spread. The chief of these agencies are dealers' stables, livery and bait stables, and horse repositories. Railway horse-boxes, and ships in which horses are carried, doubtless also play a part in the dissemination of disease. The Board of Agriculture has already promulgated Orders that are intended to counteract the dangers of infection incidental to transit by rail or sea, but it must be confessed that the measures therein prescribed under the head of disinfection are for the most part mis-named. The other sources of contagion mentioned above are infinitely more potent for evil, but they are as yet entirely ignored. On the next occasion when the Societies representative of the horse-breeding interest send a deputation to the President of the Board of Agriculture it might be worth their while to press for an extension of the regulations with regard to disinfection, and to suggest that lime-wash is not a very potent germicide.

LONDON COUNTY COUNCIL AND GLANDERS.

Exactly twelve months ago we had occasion to call attention to an interesting series of experiments and observations which were made in connection with an outbreak of glanders in the tramway stud of the Glasgow Corporation, and which furnished some fresh evidence regarding the value of mallein and the curability of the disease. At

the latter part of this number there will be found an account of the results of an inquiry which was carried out at the instigation of the London County Council, and which covered precisely the same ground as the Glasgow experiments. In London, as in Glasgow, the general plan of the experiments was to take from stables in which clinical cases of glanders had recently occurred a number of horses that had reacted to mallein, to subject these horses to repeated tests with mallein, and to observe—(1) whether any of them subsequently became clinically glandered; (2) whether those that remained apparently healthy continued to react or ceased to react; and (3) whether those animals that ceased to react had in reality recovered from the disease. It will be observed that there is a startling discrepancy between the results obtained in the two sets of experiments. The general outcome of the observations made in Glasgow went to show that of the apparently healthy horses that react to mallein only an insignificant proportion continue to react for any considerable period, or become the subjects of clinical glanders. In the London experiments, on the other hand, a very important proportion of the reacting, and at the outset apparently healthy, horses developed clinical symptoms of glanders, and a still larger proportion continued to react to mallein for a long time after the initial test. The total number of the Glasgow horses that reacted to the first test was 278, and of these only three became clinically glandered during the next nine months. In the London experiments sixty-three horses reacted to the first test, and of these no fewer than thirteen became clinically glandered and had to be destroyed, while seven others were proved by cultural or inoculation experiments to be glandered when they were killed at variable intervals after the first test. Nor does this statement fully disclose the extent to which the results of the London experiments were less satisfactory than those obtained in Glasgow, for in the former series, in addition to the thirteen horses that became clinically glandered and the seven others that were proved to be glandered by *post-mortem* experiments, nine animals were killed and pronounced to be glandered on the evidence afforded by an ordinary *post-mortem* examination. It is only fair to observe, however, that some of these nine horses had either ceased to react, or latterly had had an indecisive rise of temperature, and may, therefore, have recovered although glanders nodules were detected in their lungs; but, on the other hand, the majority of them had reacted distinctly to the last test before they were killed, and were, therefore, presumably still the subjects of active glanders. It may fairly be said that almost the half of the London horses suspected on account of a reaction to mallein developed external symptoms of glanders, or were proved not to have recovered from the disease during the course of the observations, whereas the interpretation that was placed upon the Glasgow experiments was that 275 out of the 278 horses made a com-

plete recovery from glanders. The report regarding the London experiments is accompanied by a table showing for each horse the maximum rise of temperature and the size of the local swelling on the occasion of each test, but we have not been able to reproduce this on account of want of space. A perusal of it shows that in several cases cultural or inoculation experiments made after the animal's death proved that it was actively glandered, although on one or more occasions it had failed to react to mallein. It must, therefore, be taken as certain that when the mallein test is repeated at such short intervals as a month it is not safe to regard non-reaction as conclusive evidence that the horse is no longer the subject of glanders.

It is by no means easy to understand why the Glasgow and the London experiments have yielded such very different results. It might be suggested that some serious error must have been allowed to creep into the Glasgow experiments, and that possibly a large number of the 278 animals that were supposed to have reacted to mallein never were the subjects of glanders. Opposed to that suggestion, however, is the fact that out of 93 apparently healthy horses that were condemned in the same stud because of a supposed reaction to mallein, 81 were found to present lesions of glanders when they were killed soon after they were tested. Hence, although it is possible that in a few cases an accidental rise of temperature was regarded as a reaction, there does not appear to be any reason to suppose that such errors were numerous, or that any considerable number of the 278 horses condemned as glandered were free from the disease. And yet at the end of four months after the first test 275 of these animals had ceased to react, and nearly a year after the first test they were apparently healthy! Assuming that during the past twelve months there has not been any recrudescence of the disease in the Glasgow Tramways' stud, and that the 275 horses supposed to have recovered from glanders have not within that period developed any clinical symptom of the disease, one can only congratulate the Glasgow Corporation upon what appears to have been an exceptionally favourable result of the method employed to get rid of the disease, and lament that the first attempt to profit by the example should have proved so unsuccessful.

As is shown in Mr Spencer's report, the method of dealing with outbreaks of glanders that was adopted in these experiments is a very economical one, at least as regards the first cost, but nevertheless it cannot be recommended for general adoption if no better results are to be expected from it than were obtained in London. In considering the relative merits of this method of attempting to deal with glanders, and of the alternative plan of insisting upon the application of the mallein test to all reasonably suspected horses, and the compulsory slaughter of all reacting animals as well as of those clinically affected, more must be taken into consideration

than the initial cost to the Local Authority. The two methods are not strictly comparable, because, whereas the second of them would almost certainly soon lead to the complete eradication of the disease, there would be no such certainty that the same result would be obtained by the first method. When in the case of any given outbreak the whole stud has been tested, and all the clinically affected and the reacting animals have been killed, it is easy to calculate the cost of dealing with that outbreak, for the initial expense is the only one. But who can say what may be the ultimate cost of dealing with an outbreak in which the reacting animals are left alive, and subject to no restrictions except that they must not be stabled with healthy horses, and must not be sold except to the knacker? The degree of supervision that would be necessary to obviate the risk of infection from these semi-isolated animals in a large town is practically impossible, and it is highly probable that in some cases they would be the means of spreading the disease to fresh studs. Besides, it must be remembered that the cost to the owner as well as the expense to the Local Authority must be taken into consideration in this connection. If one might accept the Glasgow results as typical of what is to be expected from the method that was there employed, that system of dealing with outbreaks would obviously be most economical to both owner and Local Authority, but if things are likely to work out as they did in London many owners would doubtless prefer to have the reacting animals promptly slaughtered, even if only quarter value were paid in compensation.

There can be no question that in dealing with isolated outbreaks outside of London and a few other large towns the wisest plan is to apply the mallein test to all the horses in the infected stable, and to insist upon the prompt slaughter of every animal that reacts, reasonable compensation being, of course, allowed to the owner. The great difficulty in the way of adopting this method in London lies in the large number of studs that are already more or less affected with the disease, and the great initial expense that would have to be incurred in the way of compensation. It is certain, however, that the flux of time will not lessen this difficulty, and that the present method of temporising with the disease cannot be continued indefinitely.

ABORTION IN MARES.

REPORTS which have reached us from different parts of England and Wales prove that during the present year cases of abortion among mares have been exceptionally frequent. In some localities the number of mares foaling prematurely has been so great as almost to warrant the use of the term "Epizootic abortion," although, in the

absence of distinct evidence of contagion or infection as a factor in the case, its employment is admittedly inadvisable.

If one may judge from the scanty references to the subject in veterinary literature, abortion among equine females has hitherto seldom occurred in circumstances that pointed to contagion as the cause. In an article which appeared in a former number of this *Journal* (Vol. VII., p. 362), Professor Penberthy stated that certain facts that had come under his notice had led him to believe "that enzootic abortion among mares is contagious, and that contagion is often conveyed from affected to healthy situations by mares which have aborted, and from mare to mare in breeding establishments, by attendants, etc.; also that places may become and remain for some time contaminated by the residence of aborting mares, etc." This opinion was supported by an account of a few cases in which the introduction into a breeding stud of a mare, or mares, that had recently aborted was soon followed by the abortion of a considerable number of the pregnant mares belonging to the stud.

We have referred to the subject here mainly with the object of inducing veterinary surgeons who may be in the possession of similar evidence, or indeed of any evidence at all, bearing on the cause of abortion among mares to be good enough to send us an account of their observations. It is perfectly certain that if contagion is the ordinary cause when a large number of mares abort in the same locality or stud, the ascertainable facts do not always make this clear. The same may, of course, be said regarding occasional outbreaks of diseases that are universally admitted to be spread by contagion, and by contagion only; but in the case of such diseases the operation of contagion is often obvious and never certainly excluded by the circumstances. It would perhaps be going too far to say that it is common to find outbreaks of abortion among mares in circumstances that exclude the possibility of contagion, but, on the other hand, we are inclined to think that the evidence pointing to contagion as the starting-point of an outbreak is seldom so strong as to be convincing.

In most of the cases that have recently been brought under our notice the act of abortion was accompanied by most serious constitutional disturbance on the part of the mare, and in not a few cases death occurred within a day or two after the foal was expelled. In this respect the disease stands in marked contrast to the so-called epizootic abortion among cows, in which, as is well known, the foetus is generally expelled with abnormal ease, and without any serious disturbance of the general health of the cow. In the mare there is a marked tendency to metritis of a very alarming character after the foal has been expelled, and also to pneumonia and laminitis. Moreover, serious symptoms on the part of the mare, including a high temperature, are sometimes the first indication that abortion is

impending. The foal is generally dead, but perfectly fresh, although the metritis that follows is usually of a putrid type. The course of events is certainly such as to suggest to the mind a bacterial invasion of the womb, determining the death and expulsion of the foal, and after that a severe attack of metritis, with, sometimes, metastasis to the lungs or feet.

It must not be forgotten that even on the view that the essential factor in the causation of the abortion is a bacterial invasion of the womb, it does not follow that every outbreak has contagion for its starting-point. It is conceivable that the first of a series of abortions in the same stud may have had a sporadic origin, that is to say, the offending bacterium may previously have been leading a saprophytic existence in the soil or elsewhere outside the animal body. The first case might thus be independent of any antecedent one, although it served as the starting-point of an outbreak in which all the subsequent cases were due to contagion. However, as we have already said, we write with the object of collecting from those who possess it the information necessary to guide to a correct conclusion regarding the etiology of the disease.

Reviews.

Walley's Guide to Meat Inspection. Fourth Edition. Re-written and enlarged by Stewart Stockman, M.R.C.V.S., Professor of Pathology and Lecturer on Hygiene and Meat Inspection, Dick Veterinary College, Edinburgh. Edinburgh and London: Young J. Pentland, 1901.

As is well known, the late Professor Walley was an enthusiast on the subject of meat inspection, and he ranked as the first authority on it in this country. The first edition of his text-book laid down for the first time in the English language the principles upon which rational and scientific meat inspection ought to be conducted. The ready sale which this and the succeeding two editions met with may be accepted as proof of the usefulness of the book, but without any disparagement of the work as it originally appeared it may be said that the present edition is greatly superior to its predecessors. The original matter has been re-written, expanded, and brought up to date, and a large number of illustrations have been added. All the abnormalities which are likely to be encountered in the bodies of the animals slaughtered for food purposes are dealt with, and at a length proportional to their importance from a hygienic point of view. As might have been expected, tuberculosis comes in for a large share of attention, and a most excellent account is given of the pathology of the disease, and of the principles that ought to guide the procedure of a meat inspector in dealing with the carcasses and organs of tuberculous animals. The work cannot fail to enhance the Editor's reputation as a teacher and an author, and we can strongly recommend it to students and practical meat inspectors.

A Text-book of Veterinary Medicine. By James Law, F.R.C.V.S., Director of the New York State Veterinary College, Cornell University. Ithaca : Published by the Author, 1901.

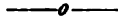
PROFESSOR LAW, whose name is well known in veterinary circles on both sides of the Atlantic, has undertaken the task of writing a text-book covering the whole field of the practice of veterinary medicine at the present day. It may almost be said to be a Herculean labour, and even those with high qualifications for the work might be pardoned for hesitating about undertaking it. As a teacher Professor Law was no doubt stimulated to make the attempt by the absence of any really scientific text-book on the subject in the English language, for, if we except the American translation of the standard work of Friedberger and Froehner, it may certainly be said that there is no English text-book of veterinary medicine that is quite abreast of the times.

The complete work is intended to run to four volumes, and three of these have now been published. The first is mainly devoted to the diseases of the respiratory and circulatory symptoms, the second to the affections of the digestive organs, and the third to the diseases of the nervous system, genito-urinary organs, eye, and skin, and constitutional diseases. The fourth volume, which is now in preparation, will treat of the parasitic and infectious diseases, and of sanitary science and police. When it is added that it is not merely the diseases of the horse that are described under these heads, but that the internal ailments of all the common domesticated animals receive consideration, some idea may be formed of the labour involved in writing such an encyclopedic work.

We must frankly express our doubts as to the wisdom of any single individual attempting to produce a text-book professing to cover the whole ground of the practice of modern veterinary medicine, for it is clearly impossible that any human being can have had the opportunities necessary to enable him to speak with authority on all the diseases of all the domesticated animals. A work that is produced in that way must necessarily be of very unequal merit in its different parts, and the present book is no exception to the rule. Another defect in the work is that in consequence of the subdivision of diseases which has been adopted there must necessarily be a certain amount of over-lapping and repetition. For example, when all the diseased conditions of all the different systems of organs have been described, it becomes impossible to give a complete account of the infectious and bacterial diseases without indulging in needless repetition.

But when we have made these adverse criticisms, which touch mainly the scope and plan of the work, there still remains much that may be said in commendation of Professor Law's text-book. It deserves to be described as a scientific as well as a practical treatise, it contains an immense volume of material presented in very readable language, and on many subjects it embodies opinions that are founded on a wide experience. In a sense it is the only book of its kind in the English language, for there is certainly no other in which the practice of veterinary medicine is discussed from the present-day stand point of morbid anatomy and pathology.

CLINICAL ARTICLES.



CASES OF ŒSOPHAGOTOMY IN THE DOG AND CAT.

By G. MAYALL, M.R.C.V.S., Norwich.

Case I.—On the evening of 7th July a fox terrier dog was brought to the infirmary with the history that he was thought to have a bone in his throat.

There was dribbling of saliva from the mouth and frequent swallowing movements. I opened his mouth and examined the palate and pharynx, but could find nothing. On feeling down the œsophagus and pressing on one side of its course, with my left hand against the fingers of my right hand, I detected a hard substance about half-way between the pharynx and where the gullet enters the thorax. This substance was not easy to detect, as the dog's throat was very fleshy. I wrote a letter to the owner of the dog, telling her I detected a foreign body and asking her permission to operate, at the same time stating that the operation might be serious, but it was the only hope. The lady gave consent to operate.

Next day the dog was put on the operating table on his back. His neck was shaved. A piece of tape was fastened round his bottom jaw behind the canine teeth, and his neck well stretched. The foreign body was with some difficulty again located. On cutting through the skin the jugular vein was exposed and pushed aside, and by pressure on the opposite side of the throat the spike of a piece of bone was brought into view. This spike was grasped with a pair of forceps and pulled out. The œsophagus was detached as little as possible from its surroundings, and through an incision about half an inch long a triangular piece of bone was removed. This piece of bone was about two inches long, and not quite half an inch broad at its base, and it was the point of the triangle we first brought into view. It appeared to be a splinter off the shaft of a leg of mutton bone. Two catgut sutures were put through the coats of the œsophagus, and three silk sutures through the external wound. The wound was dressed daily, from the 8th till the 23rd July, with chinisol solution and boracic acid powder. The sutures were removed on the 11th. There was some discharge from the wound, and occasionally curdled milk came through the opening.

The dog was fed after the operation on Brand's essence and milk with boracic acid in it. On the 23rd he was discharged perfectly cured, and was then eating solid food cut and broken up small.

We did not give chloroform, as without it the running movements of the dog, when pressure is exercised over the region of the foreign body, assist in its location. If the foreign body is small or lying

lengthways in the œsophagus it is not easy to detect. Chloroform stimulates the mucous membrane of the throat, causing a flow of saliva which may cause fresh movement of the foreign body before or during operation.

Case II.—A cat was brought to the surgery with the history that it had got a fish bone in its throat. We stretched it on the operating table and gave it chloroform, as cats are troublesome animals unless under perfect control. We fastened a piece of string to the lower jaw, and stretched the neck well out. We shaved the hair off throat. Previous to giving chloroform we located the position of the bone by the cat wincing and evincing pain on pressure over its seat.

We cut down on this spot, and felt a piece of fish bone. There was a good deal of hæmorrhage, and we were a long time grasping the fish bone. Eventually the bone broke in two, and we only got one half of it. The chloroform caused a great deal of salivary discharge, and probably this assisted the cat in swallowing the other half of the bone. The patient was under chloroform twenty minutes.

We sutured the œsophagus, leaving the external wound open. There was a good deal of discharge from the wound, which was dressed with chinisol solution.

The lady took her cat home on the fifth day after operating, and the wound was then discharging. I gave instructions that the cat was to be kept on liquid food, and sent a bottle of chinisol solution and some sal alembroth wool to dress the wound with.

Three months afterwards I met the lady and asked her how her cat was. She said it was all right, and had done remarkably well after leaving the infirmary.

Case III.—On the 13th September I was called to see a wire-haired fox terrier puppy dog. Found puppy very lame in region of near shoulder. It shrieked with pain when touched there. Gave a purgative pill and ordered warm fomentations to the shoulder, and left soap liniment to rub the shoulder after fomentation. Attended puppy until the 28th September, at which time the lameness and pain had completely disappeared. On 22nd November the dog was brought to the surgery with an inch of the pointed end of a bonnet pin sticking out of its abdomen on the left side, about an inch to one side and the same distance behind the point of the sheath.

The dog was put on the operating table and chloroformed, and the point of the pin grasped and pulled, and it came right out minus the head. Apparently the material fastening the head to the shaft of the pin had become dissolved by the gastric and intestinal juices, and had been parted with some time before the point of the pin made its exit. The external wound was dressed with iodoform collodion.

The dog made a perfect recovery, and to-day has grown to twice its original size, and is quite well and happy. Nobody saw the puppy swallow the pin, but I associate the lameness with the ultimate appearance of the pin.

POISONING OF CATTLE WITH FEED-CAKE.

By H. CAULTON REEKS, F.R.C.V.S., Spalding.

THE many varieties of "feed-cake" now on the market for the fattening of cattle point conclusively to their value for that purpose. Their popularity and demand, however, together with the competition existing between manufacturers, will be enough to show the observer that they are articles which will be extremely likely to become adulterated with matters decidedly detrimental to the proper feeding of stock. The maker attempts to turn out a commodity at a cheaper rate than his business rival. The agriculturist too often is tempted to buy in the cheapest market. The end result necessarily spells adulteration. This adulteration, so long as nothing really harmful is added, affects only the farmer from a view of economy. When, however, there are included substances giving rise to other results than the mere loss of feeding properties the veterinarian becomes interested, in that his diagnosis in certain other cases may become considerably affected.

A casual inspection of one of these feed-cakes, as now manufactured, shows it to be composed of various materials of well-known dietetic value—*e.g.*, wheat, oats, cotton, linseed, bran, and meals of different kinds, with the addition of locust, fenugreek, etc., as condiments.

Cakes composed of such ingredients, provided they have been bought in a state of purity, and are undamaged by bad harvesting, water, or fire, will, of course, answer their intended purpose admirably. Are they likely, however, to contain in addition anything absolutely injurious or poisonous? The relation of the few following clinical notes will, I think, point to the fact that they are.

My attendance was requested at a neighbouring farm to examine two cows said to be ailing. I received the following account from the owner:—

History.—There were in all forty-two animals. Inside, in one hovel, tied up for feeding, were seven beasts, and four cows suckling calves. Outside, in two yards, were twenty-seven in-calf heifers and four store steers. Preceding the date of 21st November 1900, these animals were all in good health, and were being fed with materials grown on the farm—*i.e.*, oat, wheat straw, and roots. The only foreign matter they were having was a few pounds per diem of linseed cake of a well-known brand, and presumably beyond suspicion.

21st November.—On this date the owner received the first consignment of a particular brand of "feed-cake," and commenced using it for all his stock. Those tied up in the hovel, already three parts fat, received 8 lbs. per diem; the stores and in-calvers in the yards receiving only 5 lbs. per diem.

8th December.—One of the beasts in the yard was noticed ailing, and exhibited the following symptoms: Great stiffness of the limbs

when walking, staggering gait, rapid and extreme loss of flesh, total blindness, partially fixed jaws, and dribbling of saliva from the lips. Bowels slightly constipated, complete loss of appetite, cessation of rumination, and other general signs of ill health. This case the owner treated himself with a laxative dose of medicine, and the animal recovered, save, of course, the amount of flesh it had lost.

27th December.—A second consignment of cake, of the same brand and from the same makers, was delivered. This was used as before. Between this date and 4th January 1901 two other animals in the yards were affected in a similar manner, and were also treated by the owner as the first. One other in the yard was affected in slighter degree, and treatment was deemed unnecessary.

4th January 1901.—I was called in, received the history I have related, and found an animal showing the under-mentioned symptoms:—

Pulse very slightly quickened, temperature 102.6° F., the muscles stiffened and contracted, reminding one forcibly of an acute case of tetanus, eyes amaurotic, jaws partly closed, saliva running in strings from the mouth, and a peculiar catching noise now and again in the breathing, as though the animal were choked. The bowels were stagnant, and rumination suspended. The case offered no alarming signs; I administered a laxative, and advised the disuse of the cake.

5th January.—Symptoms abating; no others attacked.

General Remarks.—These cases offer several points of interest. They clearly indicate that in any future case where there is a possibility of tetanus being diagnosed, the veterinarian should first inquire as to the kind and quality of "feed-cake," if any, used on the farm. One is also driven to ask, "Why were those animals tied up (which were receiving a larger supply of the cake) the ones to escape its effects?" Firstly, from all I can gather, I learn that cattle are not nearly so likely to suffer from the poison I suspected as horses; and that the particular poison is very erratic in its action. Secondly, it may have been that their infinitely better condition, and the fact of their being warmly housed and bedded, enabled them up to the present to better stand against the effects of the poison. Taking into consideration their larger size and general well-being, together with their shelter from exposure, the 3 lbs. difference of cake per diem would be comparatively small. Possibly, had the cake been longer continued, they also would have fallen.

The next question is, "What was the poison?" I am inclined to think, from the nature of the cake—one where materials of all kinds are ground and crushed together—from the variability of the attacks, and from the symptoms, that the Indian pea or vetch, otherwise known as "muttars," was responsible for the illness.

The annoying feature to the veterinarian is that the cases, as cases, must end in simply forbidding the condemned cake as a diet until the illnesses have stopped. One cannot prove what one has diagnosed or surmised owing to various trade influences. An attempt on the part of the owner to claim redress from the seller of the cake would mean simply a reference to the manufacturers. They, in all probability, would deny having included Indian peas or vetch in their list of ingredients for the making of that particular cake. The unfortunate part of the business, to the man who is anxious to prove

his case, is that the manufacturers may be perfectly correct in their assertion. They may not have ground up and used Indian peas in the making of this cake. They may simply have purchased a large quantity of pea meal, which is often largely adulterated with "muttars," and utilised that. The case, therefore, although I feel quite certain of my diagnosis, must remain wrapped in a certain amount of mystery.

Another feature of interest crops up in this district owing to the fact that cake of all kinds is surreptitiously given by the farm hands to the horses. A much less quantity of the same material would give rise to the same symptoms in the horse in an aggravated form—might even cause death.

I was asked by the owner, "Is the five pounds of cake, even supposing it is all muttars, sufficient to account for the symptoms?" I must confess that I was, for the moment, nonplussed. A reference to the *Veterinary Record*, however, Vol. VII., p. 43, showed me that I might answer that question in the affirmative. If the cake were largely adulterated with the pea—assuming, we will say, that one-third of the total bulk of the cake was muttars—then those animals in the yard which were stronger and more greedy than their fellows might easily have obtained enough to give rise to the symptoms.

These few notes have been hurriedly taken down in order to warn veterinarians of what is likely to occur from the excessive use of such food, and to put them on their guard in cases of paralysis or suspected tetanus.

The case is far from proved, but I am in hopes that more may be learned from the publication of this.

THE TRANSMISSION OF PINK-EYE FROM APPARENTLY HEALTHY STALLIONS TO MARES.

By the Same.

ALTHOUGH of more frequent occurrence than is commonly supposed, this matter appears to have claimed but little notice from the veterinarian. So great, however, is the wide-spread mischief it soon causes, and so heavy is the loss that it quickly entails on owners of valuable pedigree stock, that the question of its importance need hardly be raised. I am pleased, therefore, to be able to add the history of the following cases to what has already been reported.

The first reference I can find in modern veterinary literature is by Mr Pottie, M.R.C.V.S., of Paisley.¹ Mr James Clark, F.R.C.V.S., of Coupar-Angus, again drew notice to it in 1892.²

The case I am about to describe concerns a valuable pedigree Shire stallion. In this district the season and the horse's work may be said to commence with the first day of April. At this time neither I nor the owner had the slightest suspicion that anything was wrong with the horse. Soon, however, after commencing service, he began to leave in his wake diseased mares.

What we judged to be the first outbreak was on a farm of the owner

¹ "Journal of Comparative Pathology and Therapeutics," Vol. I., p. 27.

² "Journal of Comparative Pathology and Therapeutics," Vol. V., p. 261.

of the stallion. Owing to the mild nature of the symptoms, and the fact that I was not called in until after the lapse of a week from its onset, the disease was not diagnosed. I have not, now, the smallest doubt that this particular mare suffered from pink-eye. At the time, however, I reported to the owner that I could see little or nothing the matter with her. In the light of after events I am able to judge that she suffered from an extremely mild attack; and that, when I was called in, all symptoms of fever had abated and the case in that way been missed. Prior to the first noticeable symptoms this mare had been covered by the stallion in question.

Five days after this, 19th April, my attendance was again requested at the same farm to examine a pony gelding. He had been running in the same yard with, and feeding out of the same manger, as the mare above mentioned. I found unequivocal symptoms of equine influenza, known to practitioners under other and various names, *e.g.*, pink-eye, typhoid fever, bilious fever, and epizootic cellulitis. All the symptoms, as usually noticed, were well marked, and, knowing the nature of the disease and its extreme contagiousness, I immediately wrote and informed the owner of what I had discovered. At the same time I advised him to keep the animal isolated.

My advice was too late and too insufficient to be of any use. Too late, in that the next day saw fresh cases develop at another farm. Too insufficient, in that we had not yet traced the disease to its source—the stallion, but were endeavouring to course it back to what we then believed to be its true origin—one of the mares sent to the stallion for stud purposes. This, in fact, rendered our investigations difficult from the very outset. Mares had been coming to this particular stallion from Cambridgeshire, Leicestershire, Hertford, and other counties. Naturally, we first surmised that the stallion had been acting the part of an intermediary bearer, and was in no way infectious himself. However, the clinical horizon cleared during the few following days, and by 3rd May, I had on my hands the following cases:—

17th April.—First case at owner's farm.

22nd April.—Diagnosed disease in pony gelding at same farm.

23rd April.—Outbreak at another farm of owner two miles from No. 1.

25th April.—Outbreak at still another farm of owner two miles from No. 1.

26th April.—Three more cases at farm No. 1.

27th April.—Three cases at farm of Mr J. C. L., three miles from No. 1. Also outbreak at farm ten miles from No. 1.

3rd May.—Outbreak at farm eleven miles distant from No. 1.

As in the history of the outbreak related by Mr Clark, I was now in possession of the under-mentioned facts:—

- (1) In every instance the disease first commenced with a mare covered by the stallion.
- (2) Symptoms of influenza appeared from the sixth to the ninth day after service.
- (3) It then spread to other horses in the same stable.
- (4) To my knowledge there has not been an outbreak of pink-eye anywhere in the district for at least ten years.
- (5) The mares at the time of service were healthy and not suffering from colds.

- (6) The disease occurred only on premises where service had taken place and nowhere else.

Here, the close likeness between this outbreak and that related by Mr Clark ceases. Mr Clark definitely discovered that the stallion himself had previously suffered from an attack of the disease. I have not been able to do so. All last year, and, in fact, ever since I have known him, he performed his work in a satisfactory manner, and left behind him healthy mares and foals. If the stallion had been ill at home either my father or myself would have attended him.

Had he been ill while on his rounds? Enquiries elicited the fact that he had been laid up when away from home, at the latter end of last season—June 1900. I wrote to the veterinary surgeon who then attended him, asking the nature of his attack. Here is his reply:—

Dear Sir.—The stallion you enquire about I attended last year at T . . . y. I did not take any written notes of the case, so must trust entirely to my memory.

He was very stiff, with reeling gait, and loss of appetite. The next day he had griping pains with inability to pass his urine.

At the time, I considered illness due to a chill affecting the muscles of the lumbar region. I saw no symptoms of pink-eye.—

Yours very sincerely, J. M., F.R.C.V.S.

So far then, as it is possible to show, I have proved that this stallion never suffered from pink-eye. A knotty question then remained. How did he become infective? In answer to this the owner of the horse—a sharp, shrewd, clever man of business, to whom I am largely indebted for prosecuting enquiries that I could not very well have undertaken myself—discovered the following important fact:—

The first mare that the stallion covered this season came from an infected stud in Leicestershire—a stud where pink-eye raged during the whole of last autumn until Christmas.

To sum up this case is difficult. Did this horse, at the latter end of last season, suffer from a mild attack of the disease, which attack was mis-diagnosed by the veterinary surgeon in attendance? The veterinary surgeon in question is a man of some standing and years in the profession, and I feel perfectly justified in taking his diagnosis as correct and answering “no.” Also in support of the veterinary surgeon’s decision is the fact that after this illness the animal covered several mares and left behind him no trace of disease.

Did the stallion at any time during the last ten months, that is while wintered at home, have an attack of the disease, which was not reported to a veterinary surgeon? A fairly safe argument against that supposition is the fact that this particular animal is worth anything between £1000 and £2000.

Did he contract it by coition with the mare from the infected Leicestershire stud? From the facts I have gathered, I feel forced to the conclusion that he did; and that, without giving visible signs of the disease himself.

One circumstance, however, is plain.—A stallion, to all appearances in the pink of health and condition, and without ever having had any appreciable attack of the disease himself, can convey equine influenza to other animals by the mere act of coition; and, in certain cases, in no other way. I may add that this horse, though boxed up side by

side with other stallions, has not conveyed the disease to them ; and that other horses, notwithstanding they have been boxed in standings occupied the night previous by the stallion, have remained perfectly healthy. This stallion spreads the disease by contagion only ; and is not contagious except by coition.

A CASE OF ANEURISM IN THE DOG.

By CHARLES A. POWELL, M.R.C.V.S., The Sand House,
Woburn, Beds.

THE patient, a great Dane, about seven months old, was first seen by me on the 25th of January last. It was then suffering from a bad attack of sarcoptic mange, from which it made an excellent recovery. It also had *tæniæ*, and these were expelled by vermifuges.

Until the middle of March the animal's condition was in every way satisfactory, but about this time it fell away in condition and became dull and listless. I was called in on the 23rd. An examination revealed extreme pallor of the visible mucous membranes, normal temperature, and pulse regular but weak. Tonics were prescribed, with beneficial effect as regards appetite, but the general condition did not improve.

On the 3rd of April the dog rapidly grew worse, refused all food, the mucous membranes appeared quite bloodless, and pressure over the region of the left kidney caused excessive pain. I also noticed for the first time a swelling there. The weakness at this time was extreme. Death took place on the 5th of April.

A *post-mortem* examination revealed an aneurism of the posterior aorta, close to the left kidney, which was partly embedded in it. The aneurism was quite as large as two closed fists, and had ruptured—a quantity of blood being in the abdomen. There was also a second aneurism about the size of a tangerine orange in the chest, just where the anterior aorta gives off its principal branches. The heart was smaller than normal, and the walls very flabby, whilst the walls of all the main arteries appeared thinner than normal. The other organs were to all appearance healthy.

AN INTERESTING CRANIAL CASE.

By WM. BOWER, M.R.C.V.S., and FRED. HOBDAV, F.R.C.V.S.

THE patient was a hackney filly, one year old, with a purulent discharge from a hole situated in the forehead, apparently either in the upper part of the frontal bone on the right side, or just at the junction of the frontal and parietal. This had been present for some weeks, and was supposed to have been the result of violent contact with the branch of a tree in the park in which she had been turned out.

As ordinary antiseptic treatment did not produce any beneficial result, an operation was decided upon (24th January). The filly was cast, cocaine injected, and the skull trephined at the upper extremity

of the frontal sinus, as high up as it seemed safe to go, and just below the injury, the idea being to work from below. This was, however, impossible, on account of the partition which formed the roof of the sinus. The hole in the brain case was then enlarged with bone forceps until it was of sufficient size to admit a finger, and, after cautious exploration, a piece of bone about the size of a shilling was withdrawn. This was lying on the top of the brain, the coverings of the latter not being broken. Antiseptics were again applied, and recovery was uneventful.

Abstracts and Reports.

THE CAUSE OF "WHITE SCOUR" IN CALVES.

FOR several years there has been a very high mortality among calves in the south-west of Ireland, and during the past spring, at the request of the Irish Board of Agriculture, M. Nocard paid a visit to the districts worst infected, in order that he might, if possible, elucidate the cause of the mortality, and advise as to the methods of prevention. The results of his investigation were communicated to the Société Centrale de Médecine Vétérinaire of Paris, at its April meeting, and we extract the following from the report of the proceedings.

The investigations showed that two nearly related conditions were responsible for the mortality, namely, "white scour," and a form of pneumonia. The white scour generally appears immediately after birth, and it usually ends in death in the course of a few days. On the other hand, the lung disease develops less rapidly, and the subjects of it seldom die before the eighth week.

At the outset the attempts that were made to isolate the causal organism or organisms were not successful. Microscopic examination did not show anything very distinct or characteristic. The various culture media that were inoculated all yielded a variety of microbes, whether the material was taken from the blood of the heart, the clot in the umbilical vein, the liver, the spleen, the kidneys, or the lymphatic glands. None of the species isolated were found to be pathogenic on inoculation.

On the 7th April M. Nocard had brought to him the stifle of a calf which had been killed on the previous day, after having suffered from a mild form of white scour. Microscopic examination of the exudate in the inflamed joint showed, in addition to staphylococci, streptococci, and bacilli resembling the colon bacillus, a small non-motile bacterium belonging to the fowl cholera type. Cultures of this organism were found to be highly pathogenic for the guinea-pig and rabbit. Subsequently this same organism was found in the blood of the heart, and in the clot in the umbilical vessels, in three out of five calves submitted to *post-mortem* examination. In a second series of nine calves that had been the subjects of white scour this bacillus was found in seven.

In order to remove all doubt as to the etiological connection between this microbe and white scour, it was resolved to endeavour to reproduce the disease in calves by inoculation with an artificial culture. On the 16th April last two calves were received at the laboratory, one having been born on the previous day at a farm free from white scour. The other was about a week

old, and it came from a farm on which white scour prevailed. This second calf had been ill, but it was supposed to have recovered. These two calves will be referred to as calf A and calf B, calf A being the one first mentioned.

At 11.15 calf A was inoculated intravenously with 3 cc. of the organism cultivated from the stifle joint of a calf on the 7th April. At one o'clock calf B was intravenously inoculated with 10 cc. of the same culture. On the following day (17th April) calf B was quite well, although it had appeared to be a little upset on the afternoon of the inoculation. On the other hand, calf A was found to be very ill. It was unable to stand, the temperature was subnormal, and it was affected with diarrhoea, the faeces passed being yellowish and foetid. It died on the afternoon of the 17th April at 5.45. The *post-mortem* examination showed intense congestion of all the serous membranes, and of all the viscera. There was a small amount of liquid yellowish exudate in the peritoneal, pericardial, and pleural sacs, and some ecchymoses or petechial hæmorrhages under the serous membranes. The mucous membrane of the fourth stomach was congested. The contents of the floating colon were mixed with a large quantity of blood, while the mucous membrane of this intestine was thick and friable. Peyer's patches were prominent and ulcerated, or transformed into a sort of reddish pulp, easily broken down with the finger. The lymphatic glands were everywhere hypertrophied, infiltrated with a reddish serosity, and beset with fine interstitial hæmorrhages. These lesions were especially marked in the lymphatic glands of the colon. The urine was yellowish, limpid, and albuminous. The lungs were congested, and the third and fourth lobes of the right lung were oedematous. An important fact was that the umbilical vessels were quite normal, and it was therefore concluded that the calf had beyond any doubt been free from the infection when it was brought to the laboratory. Bacteriological examination of the organs of the calf yielded the following results. The blood of the heart and the pleural and pericardial exudates yielded absolutely pure cultures of the organism that had been employed for inoculation. The culture from the pulp of the liver yielded colonies of the same organism, together with a number of colonies belonging to a different species.

The experiment has held to have exactly reproduced the acute form of the natural disease.

Calf B continued in apparent good health, showing that a first attack followed by recovery confers a very solid immunity, and proving at least a theoretical possibility of vaccinating calves against white scour. It was recognised that in practice such vaccination would be very difficult owing to the rapid evolution of the disease, and a remedy or a means of prevention had therefore to be sought in some other direction.

The nature of the disease having been thus clearly determined, it became necessary to consider what was the natural method of infection. Diarrhoea being the principal symptom, one would naturally think that the microbe must be introduced by way of the mouth. Notwithstanding this, M. Nocard has arrived at the conclusion that in the majority of cases infection takes place by way of the umbilicus. He does not believe that calves are infected before birth, but considers that the specific microbe finds entrance into the tissues of the umbilical cord through the latter being brought into contact with the soil or dirty litter immediately after the calf is born. With a view to preventing the disease, M. Nocard therefore recommends that the following instructions should be circulated among the farmers in those districts in which the disease has been known to prevail:—

“White scour is generally the consequence of an umbilical infection which takes place at the moment of parturition.

“Farmers may prevent the disease by conforming strictly to the following instructions:—

"1. Cows that are at the point of calving ought to be provided with dry and clean litter until after the act of parturition.

"2. As soon as the premonitory signs of parturition are observed the vulva, anus, and perineum ought to be washed with a warm solution of lysol, of a strength of 20 grammes to a litre of water. At the same time a large quantity of this solution ought to be used to syringe out the vagina.

"3. As far as possible the calf ought to be received into a clean cloth, or at least on a thick layer of fresh litter which has not been soiled by urine or excrement.

"4. Immediately after birth the cord should be tied with a ligature that has been soaked in lysol and has not been soiled by urine or excrement. The cord should then be cut below the ligature.

"5. The remainder of the cord and the umbilicus should be mopped with the following solution:—

Rain water	1 litre.
Iodine	2 grammes.
Potassic iodide	4 "

"6. The disinfection of the umbilicus and cord should be completed by applying the following:—

Methylic alcohol	1 litre.
Iodine	2 grammes.

"7. When the alcohol has evaporated the operation will be completed by dressing the cord and umbilicus with a thick layer of iodised collodion (1 per cent.). As soon as the collodion has dried the calf may be left with its mother."

M. Nocard is firmly convinced that if these directions are followed white scour will be prevented.

RINGWORM INFECTION IN MAN AND ANIMALS.¹

SINCE the researches of Gruby on the subject of ringworm, communicated first to the Académie des Sciences in the years 1841 to 1845, the subject has been investigated by numerous observers both in England and abroad, and our knowledge of the subject, both clinically and pathologically, has advanced very considerably. This advance is to a great extent due to the brilliant researches of Sabouraud, who since the year 1892 has contributed several valuable papers on the subject. It is now generally held that ringworm in human beings is usually due to two classes of fungus, the "microsporon" and the "trichophyton," which may be distinguished to a certain extent clinically and microscopically, but still better culturally. A less common form of ringworm is due to a fungus which culturally gives a faviform growth, but the lesions of which do not resemble favus, but are either circinate or a well-marked kerion. The trichophyton class includes two forms: the ectothrix, both large and small spored, and the less common (in England) endothrix, while microscopically an intermediate variety can be distinguished, in which the fungus lies both within and without the hair. The question of etiology of these different forms has a ways been one of extreme difficulty, for the patients are in most cases children, who, either at school or at play, may very easily have associated with other children suffering from ringworm; and, seeing how very numerous the cases of ringworm in London are, the exact source of contagion becomes almost impossible to trace. In the case of adults affected with trichophyton the source of infection may sometimes be determined with considerable exactness, and of course in children also, this is sometimes possible.

¹ Brit. Med. Journ., 1901, May 12.

During the past three years in the skin department of the Middlesex Hospital, and more recently at the German Hospital, Dr Bunch has been enabled to investigate the question of the etiology of a large number of ringworm cases.

Inasmuch as ringworm cultures obtained from man can be inoculated into animals, and will in them give rise to characteristic lesions in which the fungus can again be demonstrated and reinoculated, it might be argued that in these cases there is a possibility of the horse or dog having contracted the disease from the human being instead of *vice versa*, but this possibility can be practically eliminated by comparing the dates at which the first symptoms were noticed in the two cases.

The first case was that of a girl, aged 3½, in whom had been noticed a week previously a small ringed patch on the right forearm, just above the wrist, and a few days later a second patch on the right shoulder. When seen, the first-mentioned lesion was circinate, measuring one inch in diameter, with a slightly scaly surface and a red raised edge, which in places showed some minute vesicles containing clear serum, and the redness extended somewhat beyond the vesicular border, fading off gradually into the surrounding skin. The lesion on the shoulder was also circinate, but smaller; the surface was dry and somewhat scaly, and there was no vesiculation. Microscopic examination showed chains of large squarish spores both inside and outside the hairs, and in preparations treated with 6.8 per cent. liquor potassæ a coarse segmented mycelium was seen in the intrafollicular portion of the hair shaft, terminating in a fringe above the bulb, and also outside the hair in the root sheath and in the connective tissue of the follicle. Scrapings from the lesions showed also chains of spores crossing between the epithelial cells, and a more abundant fine branching mycelium, segmented at somewhat longer intervals, and showing in potash preparations as bright glistening threads, which were, however, less highly refractile than the square-shaped spores. In places, this mycelium gave off short lateral offshoots, which swelled out at the extremity into small oval bodies; and from these, again, there occasionally sprang a still finer and apparently quite recently developed mycelial thread. Similar mycelial appearances were observed in ectothrix preparations from other cases, and also in microsporon cases.

Cultures from minute portions of the affected hairs and from the scales, grown on French proof agar, the materials for which were obtained from Paris, so that the cultures might be accurately compared with those of Sabouraud, showed a network of coarse aerial hyphæ, radiating from the centre, white in colour, and terminating at the edge in tapering, somewhat pointed, processes.

Dr Bunch learned that the child's father was a coachman, and that there was a horse in his stable which had been certified by a veterinary surgeon to have ringworm, and that the child had been brought into contact with the horse. On paying a visit to the stable he found that there were two patches on the horse, one on the neck and one on the head, both scaly, and covered with broken and twisted hairs, but showing no sign of vesiculation. Microscopic examination of these hairs showed chains of large spores outside the hairs, just as in the child, and cultures on the same medium proved to be almost identical with those obtained from the child's scalp.

The second case was also one in which infection was derived from a horse, but the species of fungus was here quite different. The patient was an ostler, aged 19, with a circular scaly patch, about an inch in diameter, on the right side of his neck. This patch was said to have been present a week, and it began as a small spot, which spread gradually at the edges while clearing in the centre. When seen the edge was red and somewhat raised, with a few small scattered papules here and there. Examination of scales from the

lesion showed a network of abundant, fine, branching mycelium, irregularly septate. Fine lanugo hairs showed numerous round or oval small spores, and on careful focussing mycelial threads could be made out running chiefly in the direction of the long axis of the hair. The microscopic appearances, therefore, did not point to any species of trichophyton, but cultures left no room for doubt. The fungus grew with extreme rapidity, appearing first as a white downy growth, which rapidly spread over the surface of the medium. This culture closely resembled one of microsporon obtained directly from a horse by Bodin.

On inquiry it was found that the ostler had been looking after a young horse with some "pimples" on its nose, and on going to the mews Dr Bunch found that the animal had some indistinctly ringed vesicles above the right nostril, and the hairs around appeared to be irregular and broken. Microscopically they showed a sheath of small spores outside the hair, and the hair itself contained fine mycelium. Cultures gave a rapidly growing white downy mass similar to that obtained from the lesion of the patient. Microscopic examination of the aerial hyphæ from the culture showed an unusual appearance, inasmuch as the organs of reproduction proved to be lateral bud-like growths, from 2 to 3 μ in maximum length, situated directly in the mycelial threads.

The third case was that of a boy, aged 3, with kerion of the scalp. There was a patch, about $\frac{3}{4}$ in. in diameter, on the left parietal region, crusted with yellowish scabs, which had been present for a fortnight. The hairs were broken, and could be very easily removed. They mostly came away without the bulb, and the hair shaft showed a typical felting with a mosaic of small spores, while inside the hair was mycelium, terminating in a fringe above the region of the bulb. Cultures showed alternate concentric rings of white and brown growth, with a delicate filiform border and a central raised prominence. Hairs from a terrier with which the child had often played proved to be infiltrated with typical microsporon, and cultures from these hairs resembled closely those obtained from the child's scalp.

A fourth case was rather peculiar, in that the patient was only four weeks old when brought to the hospital, and had already a well-marked patch of tinea circinata, strictly localised and circular, on the left cheek. The surface of the lesion was red and somewhat scaly, with some vesicles and a few pustules at the spreading edge. Abundant large-spored fungus was found in scrapings and in the root sheath of the hair, and on cultivation it gave a somewhat coarse growth of radiating aerial hyphæ of a dull whitish colour. A cat in the house, with which the child played, had a patch on the neck, from which the hairs had almost all come out, and on getting the mother to bring the animal to the hospital it was found that the hairs over this patch were extensively affected with ectothrix fungus. Cultures showed a beautiful white growth of very coarse hyphæ, which grew much more rapidly than that from the lesion on the child, and was more downy in appearance. The medium on which the fungus from the cat was grown was of exactly the same composition as that used for the fungus from the child, but it had been freshly prepared, whereas the other had been kept a short time, and had, perhaps, got a little dry. This may account for the greater luxuriance of the growth in the second culture.

The fifth case was also one in which the source of infection had been a cat. The patient, a youth aged 16, had several well-marked patches of tinea circinata on the left arm and trunk, two of which were slightly vesicular at the edge, but the rest scaly. A large-spored trichophyton was found both within and without the hairs of the lesions, and on cultivation gave a somewhat coarse white growth very similar to what has already been described by other observers as being derived from the cat. From a patch on the patient's

cat, which had been noticed to be "mangy," a very similar culture was obtained.

The next case was rather exceptionally interesting, in that the fungus which gave rise to the infection was an endothrix, and an endothrix is supposed to have an animal origin but seldom. Indeed, this species of fungus can only be inoculated into animals with difficulty, but it can be inoculated on the guinea-pig, the rabbit, and the cat, and this being so, there can be no reason why human beings should not become infected from animals. The patient in whom this infection occurred was a boy, aged 9, who came under observation for two more or less circinate, smooth, almost bald patches on the scalp, which had been present for more than a month. The hairs were said to have come out rapidly at first, and the patches were never markedly scaly. When seen, there were scattered over the affected areas some stumps of hair, dark in colour, projecting a few millimetres above the surface of the scalp, and swollen slightly at the far extremity. Within these hairs were chains of large spores running chiefly in the direction of the long axis of the hair, but no fungus could be found outside the hairs, which came away easily, leaving the root sheath behind. The surface of these patches was so smooth that it was very difficult to scrape any of the superficial epithelium away, but in none of the *débriés* thus removed could any fungus be found. Cultures gave an irregularly plicated growth, somewhat powdery, and with a tendency to crack on the surface. Hairs from a cat in the house also contained an endothrix fungus, but the surface of these lesions on the cat was not smooth, and was covered with fine scales. Cultures from these lesions on the cat proved to be very similar to those obtained from the boy.

A case in which the nature of the fungus was open to some question was that of a girl, aged 13, with a patch of *tinea circinata* on the forearm, the surface of the lesion being scaly and nowhere vesicular, though at the margin there were some red raised papules. Both in the scales and in the hairs was present a mycelium, rather delicate, and divided at irregular intervals by transverse septa, branching freely in its course. Cultures gave a dull, opaque, whitish growth, spreading somewhat irregularly from the centre, and with a faviform appearance. The growth was quite unlike anything obtained from either trichophyton or microsporon infections, but the clinical appearances differed in no way from an ordinary *tinea circinata*, and presented no possible similarity to favus. On inquiry it was found that there was a pet canary, whose feathers had been coming out in patches, and microscopic examination of these feathers showed an invasion by a fungus similar to that already demonstrated in the patient. The cultures, too, from the bird were faviform in appearance, and only differed in that the spreading edge was slightly more thickened.

The last case was one of ringworm of the beard in a man, aged 29. On the right side of the face were two small scabbed pustular lesions, over which the hairs were broken off within a short distance of the surface of the skin, and were fixed but loosely in the follicles. There was also a single red ringed patch with a pustular edge on the glabrous skin of the same side of the neck. When the hairs were pulled out, the root sheath often came away at the same time, and on microscopic examination this was found to be packed with chains of large glistening spores, and at the mouth of the follicle a collarette of mycelium was in some instances present. But also within the hairs there was some fungus present, though there was no apparent stripping up of the cuticle. Cultures gave a whitish downy growth with a central prominence, and a fine striated edge, but in many of the original tube inoculations the growths were contaminated by staphylococci, and it was only with difficulty that a pure culture was obtained.

The patient, who was a farm labourer, stated that one of the calves had

ringworm, and brought some hairs from one of the patches. These hairs also contained an endo-ectothrix fungus, somewhat closely packed, so that the chain formation was not very evident. Cultures from these hairs also gave a white aërial growth with a striated edge.

Dr Bunch remarks that valuable though microscopic examination is in enabling a diagnosis to be made, greater reliance must always be placed upon cultures, which either confirm the conclusions derived from the microscope or serve to differentiate those cases where otherwise a differential diagnosis could not be made. Such, for instance, are those cases where the spores are so closely packed that their shape necessarily appears round, and the microscope is quite unable to distinguish between microsporon and ectothrix. Cultures, however, soon clear up the diagnosis.

The prognosis in the above cases varied very considerably, for though the inflammatory process was in most cases acute, yet it often yielded readily to treatment; in fact, some of the apparently milder cases proved in the end the most resistant.

Investigation of the organs of fructification in the trichophyton cases showed that they were either clusters of spores or conidia placed laterally on the mycelial hyphæ, or chlamydospores. Rarely intramycelial endoconidia from 10μ to 12μ in maximum diameter were found. The clusters of spores were usually borne close to the end of a mycelial thread, sometimes in considerable numbers, so as to form, as it were, a terminal inflorescence. The conidia were from 2μ to 4μ in length, and were borne either directly on the mycelium or on short unbranched pedicles. The chlamydospores or multilocular conidia were pyriform in shape, or somewhat fusiform, with a long diameter of from 25μ to 40μ , and enclosed in a capsule which did not stain with acid fuchsin. The contents were a granular protoplasmic material, divided into two or more loculi by means of transverse septa. These multilocular conidia are borne, like the unilocular conidia, either laterally on the mycelium, or at the end of short pedicles. In some cultures they are very numerous; in others they do not appear until the culture has grown for some time, and then only in small numbers as compared with the inflorescences of groups of spores. Chlamydospores are often numerous in old cultures, in which degenerated white downy growths appear, and here they seem to appear at the expense of the unilocular conidia. In the case of the microsporon derived from the horse, reproductive organs in the form of chlamydospores were also found, divided by one or more transverse septa, and containing granular protoplasm. At the end of some of the mycelial hyphæ occurred conidia somewhat cylindrical in shape, with occasionally transverse partitions dividing them up into loculi. Other reproductive organs found in these cultures were conidia borne laterally on the hyphæ in the form of bud-like processes, and measuring about 3μ in maximum diameter.

GLANDERS IN LONDON.

Report of Mr ALFRED SPENCER, Chief Officer of the Public Control Department, London County Council, as to the increase of Glanders in London, and as to experiments bearing on the curability of Glanders.

The Public Control Committee at its meeting on the 10th May directed me to report—

(a) As to the recent increase of glanders in London, and the probable causes of such increase.

(b) The measures which have been taken by the Council for dealing with the disease, and the further measures which appear necessary,

having especial regard to the recommendations of the Departmental Committee, and the experience which has recently been obtained as to the curability of glanders; and to include in the report information as to the experiments carried out to ascertain the effect of the persistent inoculation with mallein of certain infected studs in Glasgow and London.

The progress of glanders in London was last dealt with in a report from the Public Control Committee presented to the Council on the 10th July 1900. After summarising the recent history of the disease, and the recommendations of the Departmental Committee, the report drew attention to the recrudescence of the disease which commenced last year, and recommended that the Board of Agriculture be urged to take steps for giving effect to the recommendations of the Departmental Committee.

No satisfactory reply having been received from the Board, the Public Control Committee directed me to see Mr Elliott, the Secretary of the Board, and on the 14th December last I reported that Mr Elliott stated that—

“The Board are of opinion that as a preliminary to the issue of a new Glanders Order, or to altering the present procedure, it would be essential to ascertain—

“(1) Whether horses which have reacted to mallein, but which do not present clinical symptoms of glanders, are in a condition to infect other horses.

“(2) Whether there is any curative effect resulting from the repeated injections of mallein.

“With the object of determining this, the department had appointed a small committee of experts, consisting of the veterinary advisers of the Board (Mr Cope and Professor M'Call), Professor M'Fadyean, Principal of the Royal Veterinary College, and Mr Hunting, one of the Council's veterinary inspectors.

“This committee were now considering the question, and had been asked to prepare estimates as to the cost likely to be incurred by the enquiry, with a view to an application being made by the Board of Agriculture to the Treasury for the necessary funds.”

I understand that the expert committee are now carrying on their investigations, and that their inquiry will probably extend over a somewhat lengthy period.

Meanwhile the increase of glanders which commenced in London in 1900 is continuing, and as the increase has been considerable it becomes necessary to consider whether some immediate measures should not be adopted for checking it.

The numbers of cases of glanders in London reported since 1889 are as follows:—

1889	1052	1895	1036
1890	1503	1896	857
1891	2174	1897	980
1892	2520	1898	845
1893	1010	1899	905
1894	982	1900	1387

During the first four months of the present year there were 538 cases reported, a rate equal to about 1700 a year, and at the present time the rate is if anything rather higher.

It is difficult to assign a cause for this increase. There have always been considerable fluctuations in the returns as to glanders in London. For instance, the number which had fallen as low as 780 in 1885 increased to 2520 in 1892, and again fell to 845 in 1898. But the increasing use of mallein in detecting latent cases of disease may be responsible for some of

the recent increase. If when an outbreak occurs in a stud the owner has all the in-contact horses injected, and quietly disposes of any which react, he protects himself at the cost of other horse owners, and creates fresh centres of infection by spreading the disease to other stables. There is some reason for believing that unscrupulous owners take this course, and if it is practised to any considerable extent it would largely account for the increase in the spread of the disease which is now taking place.

The Council has taken, and is taking, all the measures for repressing glanders which the existing law affords, and has pointed out to the Board of Agriculture the importance of certain additional measures for the suppression of glanders, but it is powerless to prevent the sale of apparently healthy animals. The effective method of suppressing glanders is to treat all horses in an infected stud as suspicious, and only let them go free when they have been proved healthy. Practically all the experienced witnesses before the Departmental Committee were in agreement as to the proper way of dealing with the disease, and the Committee itself formulated a series of recommendations which, if adopted, would probably be successful in suppressing it.

At present there is no legal obligation on a veterinary surgeon to give notice of cases of glanders coming to his knowledge, and although the owner is under such an obligation there is reason for believing that many outbreaks of glanders are not reported. The Departmental Committee, therefore, recommend that it should be made compulsory for veterinary surgeons to notify cases of glanders which come to their knowledge, and by this means it is hoped that the great majority of outbreaks will be brought to the knowledge of the authorities. If, in addition to this, the Local Authority were empowered to deal not only with the clinically glandered cases, but with the animals in the same stud, or such of them as had been apparently brought within the sphere of contagion, there would be a good prospect of greatly reducing the disease, if not of suppressing it altogether.

The Departmental Committee suggest that the veterinary inspectors should have the power of declaring in-contact horses as suspicious, and that where this is done the owner should have the right to demand that any horse so declared should be tested with mallein by and at the expense of the Local Authority; and that if after one or more applications of the mallein test the veterinary inspector is suspicious that the horse is glandered, it shall be slaughtered, and compensation paid at a special rate. The Committee do not suggest what the special rate of compensation in such cases shall be, beyond limiting it to £25, but it is most important that the rate should be fixed. Indeed, it is hardly too much to say that the success or non-success of any new departure in dealing with glanders depends largely upon such a rate of compensation being fixed as will induce horse owners to co-operate cordially in suppressing the disease. For obvious reasons it is equally important that all Local Authorities should adopt the same rate of compensation, which should therefore be fixed by the Order.

Curability of Glanders.

Formerly glanders was regarded as an incurable disease, but the experience gained by the use of mallein as a diagnostic agent has gradually led to the opinion that a considerable proportion of the cases discovered by the injection of mallein never develop into clinical cases, but recover. It has not been conclusively proved whether the disease is arrested by mallein, or if the recovery is due to natural causes. Evidence was given before the Departmental Committee that out of 1114 horses belonging to the Great Western Railway Company which were tested with mallein sixty-seven reacted, and that out of this number only twenty-seven were killed, the remainder apparently recovering.

Since the Departmental Committee completed its labours an experiment on a still more extended scale has been carried on with the stud of tramway horses belonging to the Corporation of Glasgow. The history of that experiment has been published in great detail, so that it is only necessary to say that out of 4439 horses tested with mallein—

		278 reacted at the first test.
49	„	second test.
9	„	third test.
5	„	fourth test.
2	„	fifth test.
1	„	sixth test.
0	„	seventh test.

and that only three of the horses became clinically affected with glanders, and the remainder returned to work apparently recovered. In view of these results it appeared to be desirable that experiments in various studs in London should also be carried out if owners could be induced to undertake them, and ultimately arrangements were made with five owners for such experiments to be carried out.

Stud A.

This is a stud belonging to one of the large brewery companies, and an outbreak of glanders having occurred in the early part of 1900, it was arranged to have the whole stud of 164 horses tested by Mr Hunting, F.R.C.V.S., one of the Council's veterinary inspectors, and who was subsequently appointed by the Board of Agriculture as one of the committee of experts on glanders. Of the 164 horses tested twenty-one reacted at the first test, and were placed in an isolation stable and periodically re-tested from time to time with the results shown in Table A.

It will be seen that one horse (Emblem) developed clinical symptoms after the third test, and another (Bobs) after the fourth test. Another horse (Chesterfield) continued to react up to the ninth test. It was then slaughtered and found to be glandered. Of the remaining eighteen horses five were killed, and on *post-mortem* examination old glanderous lesions were found in the lungs, but in no case was the disease active, as only negative results followed the bacteriological examination subsequently made by Professor M'Fadyean, Principal of the Royal Veterinary College. The remaining thirteen horses were declared to be healthy, and resumed their ordinary work, and three of the horses from this stud were subsequently transferred to the expert committee appointed by the Board of Agriculture. So that out of twenty-one horses which reacted to the first test, two only became clinically glandered, one which continued to react proved actively glandered on *post-mortem* examination, and the remainder gradually ceased to react and recovered. In the case of the horse Chesterfield, Professor M'Fadyean states that *post-mortem* appearances of the lungs indicated that although the horse was actively glandered there were signs of a curative process in progress as several of the glanderous nodules had become calcified, and others were in a caseous state.

Stud B.

This is one of the studs of one of the large omnibus companies, and on an outbreak of glanders occurring in May 1900 it was arranged to test the whole of the stud of 349 horses, and to isolate the reactors, twenty-six in number, or nearly 8 per cent. The results of the various tests are shown in Table B.

It will be seen that out of the twenty-six which responded at the first test, six subsequently developed clinical symptoms, eleven were killed and proved to be actively glandered, two were passed on to the Board of Agriculture

Committee for further experiments, and the remaining seven were killed and proved to be no longer actively glandered, although the *post-mortem* examination revealed glanderous lesions in the lungs which had become inert.

Studs C, D, E.

These are three smaller studs, and may be conveniently grouped together.

C stud consisted of only seven horses, of which two reacted. One of these was killed after the fifth test, and the other ceased to react after the fourth test and was declared healthy.

D stud consisted of sixty-nine horses, ten of which reacted. Of these, three developed clinical symptoms, four were killed and proved actively glandered, two were killed, and though glanderous nodules were found in the lungs the bacteriological examination gave negative results; the remaining horse was transferred to the Board of Agriculture.

E stud consisted of sixty-one horses, of which four reacted to the first test. Of these, two developed clinical symptoms, and the remaining two have been declared healthy.

The details of the test are shown in Table C.

The general result of the experience gained in dealing with these five studs of horses may be briefly summarised as follows:—

Summary of Results of Experiments with Isolated Reactors.

<i>Stud.</i>	<i>Number of horses tested.</i>	<i>Number of horses which reacted to the test.</i>	<i>Number of reacting horses which subsequently developed clinical symptoms.</i>	<i>Number of isolated reactors which ceased to react on final test and were declared healthy.</i>	<i>Number of horses which were slaughtered at end of experiments, and which proved glandered on post-mortem. (No cultural experiments performed.)</i>	<i>Number of horses which proved doubtful on final test and were slaughtered.</i>	<i>Number of horses which gave negative results to cultural experiments.</i>	<i>Number of horses which on cultural experiments proved actively glandered</i>
A stud .	164	21	2	13	—	6	5	1
B stud .	349	26	6	—	5	13 2 disposed of to B of A	7	6
C stud .	7	2	—	1	—	1	1	—
D stud .	69	10	3	—	4	2 1 sold to B of A	2	—
E stud .	61	4	2	2	—	—	—	—
5 studs .	650	63	13	16	9	25	15	7

=63

The nine slaughtered at termination of experiments (col. 5) had been subjected to few tests: the first five, three tests; the remainder, two tests.

The results of the London and Glasgow experiments may be compared, as follows:—

	<i>Glasgow.</i>	<i>London.</i>
Total horses tested	4437	650
„ reacted at first first	278	63
„ recovered	275	30
„ not recovered	3	30
„ still under experiment	—	3

For comparative purposes it would have been preferable to have had a record of the reactions of the Glasgow test, so that the degree of intensity of the various reactions could be noted.

If the London experiments had confirmed those at Glasgow there would have been the strongest possible inducement to attempt to deal with glanders by way of cure whenever the affected horses could have been dealt with in an isolation stable in such a way as would have prevented risk of spreading the disease. But it is questionable whether a recovery rate of about 50 per cent. would be a sufficient inducement to horse-owners to undertake the trouble and expense of dealing with infected studs by way of repeated injections, until either clinical symptoms developed or the animals ceased to react. The experiments carried out in the five London studs were all under favourable conditions and under the close supervision of exceptionally qualified and experienced veterinary surgeons. But it was only after many months that definite results could be deduced, and in each case the owner became impatient and desired either that the horses should be slaughtered or declared free. In the case of the omnibus stud the former course was adopted, and out of twenty-six horses in the isolated stud of reactors no less than seventeen proved to be actively glandered and two continued to react. This was after a period of treatment by injection extending over eight months, and as the omnibus company continued to get outbreaks in their main stud on the same premises, they determined to have all the horses in the isolation stud killed.

As a means of comparison of the cost to the County, the actual cost of the recent experiments in five stables is set out below, together with the cost that would have been incurred if the horses had been killed after the first reaction and either quarter value or half value paid.

Stud.	Number of reactors.	Full value of reactors.	Number alive at termination of experiments.	Actual cost of experiment to Council.	Probable cost to Council if compensation at half value had been allowed, including cost of destruction of carcasses and veterinary inspector's fees.				Probable cost to Council if compensation at quarter value had been allowed, including cost of destruction of carcasses and veterinary inspector's fees.				
					£	s.	£	s.	£	s.	£	s.	
A	21	591	13 (declared healthy)	Compensation (8 horses) Destruction of carcasses . Inspection	118 16 20	10 0 0							
							154	10	342	10	194	15	
B	26	951	2 (transferred to Bd. of Ag.)	Compensation (24 horses) Destruction of carcasses . Inspection	209 48 25	15 0 0							
							282	15	532	10	294	15	
C	2	28	1 (declared healthy)	Compensation (1 horse). Destruction of carcass . Inspection	2 2 5	0 0 0							
							9	0	20	0	13	0	
D	10	391	1 (transferred to Bd. of Ag.)	Compensation (9 horses) Destruction of carcasses . Inspection	85 18 16	15 0 16							
							120	11	218	0	120	15	
E	4	145	2 (declared healthy)	Compensation (2 horses) Destruction of carcasses . Inspection	15 4 5	0 0 5							
							24	5	82	10	46	5	
	63	2,106	19		£591	1			1206	0	669	10	

It is clear that in these cases the immediate cost to the county has been less than it would have been if either quarter or half value had been paid as compensation, but it is by no means certain that attempts to effect a cure will prove cheaper in the end. Even in those comparatively few cases where the local circumstances enable something like complete isolation to be provided, a local authority would hardly feel justified in consenting to reactors working in the streets until it has been demonstrated that there is no danger of infection until clinical symptoms appear. At present that is not the case, and the Departmental Committee appear to be fully justified in the recommendation "that horses which react to the mallein test should be considered as possible sources of infection." Under frequent tests with mallein, especially at short intervals, a horse may cease to react and yet remain glandered. The expert Committee of the Board of Agriculture is investigating the question whether reactors which do not show clinical signs of glanders can affect other horses, and whether there is any curative effect from repeated injections of mallein; but although the conclusions which the expert Committee may arrive at will guide both local authorities and owners in the policy to be pursued in dealing with affected studs, the Committee's conclusions will probably not in any way influence the amendment of the law, and it does not therefore appear to be necessary or desirable to further postpone the adoption of the Departmental Committee's recommendations, especially having regard to the necessity for obtaining additional statutory powers for requiring compulsory notification.

It will be convenient if I set out in a concise form the alterations in the practice of dealing with glanders which I consider essential to success.

Summary of Conclusions and Recommendations.

1. Compulsory notification by veterinary surgeons.

Upon this point the evidence before the Departmental Committee was practically unanimous. There is little doubt that such a requirement is essential to the successful treatment of glanders. The Board of Agriculture state that a statutory enactment is necessary to give effect to it, and if this be so, it is important that the Board should introduce a Bill at the earliest opportunity.

2. Appointment of veterinary inspectors who are not in private practice as veterinary surgeons.

The Council has pledged itself to make this alteration when compulsory notification is made law. I have for many years past advocated the change, and I venture to doubt whether it would be possible to successfully deal with glanders in London while the present system of employing veterinary inspectors in private practice is continued.

3. That on an outbreak of glanders in a stud, all the horses brought within the sphere of contagion should be declared suspicious and not allowed to be moved.

This is the course recommended by the Departmental Committee, and is practically the only one which gives promise of success in suppressing the disease. At present the veterinary surgeon advises the owner to have the in-contact horses tested with mallein, but only in a *small* proportion of cases will he consent. Where he does the reacting horses are declared to be glandered and killed, and quarter-value is paid as compensation. It is obvious that it is not to the owner's pecuniary interest to adopt this course. If a horse is worth £40 the loss to the owner is £30. If the owner took the alternative course of privately injecting his horses, and afterwards selling the reactors, the loss in a horse of the value of £40 would probably not exceed £10. It is believed that in a large proportion of cases the owner adopts the course involving the least pecuniary loss, and that this is the chief cause of the increasing prevalence of glanders.

4. That the owner should have the right to demand that any horse detained or suspected shall be tested with mallein by and at the expense of the Local Authority.

It would clearly be to the owner's interest to have suspected horses tested, so that if they did not react they might be liberated. Where they did react they should either be killed and compensation paid, or they should be kept under supervision and worked as an isolated stud. After experiencing the difficulties entailed in the latter course, I am inclined to view that the better plan would be to slaughter all horses that give the distinctive reaction to two or more tests made at intervals of at least fourteen days.

5. That compensation in clinical cases should be as now, £2 (carcase value), but that compensation in cases discovered solely by the injection of mallein should be at a higher rate, but not exceeding in any case £25.

The Departmental Committee avoid recommending a rate of compensation. There would be obvious advantages if the rate were fixed by the order itself, and not left to the discretion of Local Authorities, as otherwise there will always be the risk of suspected animals being moved into the districts of Local Authorities who give the highest compensation. There is a very general opinion that if half-value were given in mallein cases owners would co-operate with Local Authorities, and the disease be largely if not entirely suppressed.

6. That the slaughter of all animals showing clinical symptoms of glanders should be made compulsory.

It is also very desirable that the experimental research now being carried out by the Board of Agriculture Committee should be completed, but whatever the result it will probably make no difference in the conclusions and recommendations above set out.

I therefore suggest—

That the President of the Board of Agriculture be requested to receive a deputation from the Council to urge the Board to take immediate steps for giving effect to the recommendations of the Departmental Committee made in 1899.

INFECTION OF CATTLE WITH ANTHRAX BY MEANS OF LINSEED CAKE.

A CASE of much interest to farmers, veterinary surgeons, and cake manufacturers was tried on the 16th, 17th, and 20th May last, in the King's Bench Division of the High Court of Justice, London, before Mr Justice Mathew and a special jury.

The plaintiff was Lord Brougham and the defendants Messrs Pattinsons & Winter. The plaintiff claimed damages for the loss of six valuable shorthorn cattle, which, it was alleged, were infected with anthrax by means of linseed cake purchased from the defendants. Mr Jelf, K.C., and Mr Shearman appeared for the plaintiff, and Mr Pickford, K.C., and Mr Willes Chitty for the defendants.

Evidence for the Plaintiff.

MR JOHN WESTGARTH, *examined by Mr Shearman*, said that he was the farm bailiff to the Plaintiff, Lord Brougham, a position which he had held for the last nineteen years. Lord Brougham has one farm with three separate buildings for these cattle, near Penrith. He breeds pedigree cattle at all three farms. They are called the Eamont Bridge Farm, the Gardens Farm, and the Hospital Farm. On the 25th March, a cow, Lady Oliva, died at Eamont Bridge. On 27th March, another cow, Langton Cherry, died at the Gardens Farm. On the 29th, a bull died at the Eamont Bridge Farm, and on

the 30th, another bull at Eamont Bridge. On the death of the first two bulls, on 9th March, he called in a veterinary surgeon to ascertain the cause of death. The surgeon was called in after death. They had been taken ill and died almost at once. He had not sufficient knowledge to know of what they died. The veterinary surgeons who were called in on the 9th March were Messrs Pears and Bowlas of Penrith. They certified on the same day that death was due to anthrax. This came from both Mr Pears and Mr Bowlas. When there is a death from anthrax it is necessary to give notice to the County Authorities. They came that night to inspect. On the 10th, the two bulls were buried 7 feet deep in quick lime, about 100 yards from the byres. They were buried on a piece of ground which was fenced round so that no cattle could get at it. The place where the two bulls died was disinfected and the straw burned. Lord Brougham had about 100 head of shorthorn and a lot of non pedigree cattle as well at these farms. The most of these were kept at Eamont Bridge. Part of the cows were kept at Gardens Farm and part at Eamont Bridge. Young pedigree cattle are kept at the Hospital Farm. Before 9th March 1890, the cattle at the Eamont Bridge Farm were fed upon hay, roots, crushed oats, Indian meal, pea meal, bran, and cake—feed cake and linseed cake mixed. The peas and so forth were purchased from a local miller. The cattle at Gardens Farm were similarly fed, but had feed cake only, until the 21st March, when they had linseed cake. Until that date no linseed cake had been supplied at the Gardens Farm. After the veterinary surgeons had certified the death on the 9th to be due to anthrax, he suspected the square feed cake. He stopped giving it to the cattle at Eamont Bridge on the 10th, and at Gardens Farm on the 21st. After the 10th, the only artificial food given at Eamont Bridge was the linseed cake. Square feed cake was used at Gardens Farm up to the 21st, when linseed cake was substituted. It was at Eamont Bridge that the cow, Lady Oliva, died on 25th March. Langton Cherry the 11th died at Gardens Farm on the 27th. The veterinary surgeons were again sent for, and certified death due to anthrax. The cow that died at Gardens Farm was buried in the park, near to the Hall. The cow that died at Eamont Bridge was buried about a quarter of a mile from the house, 7 feet deep in lime, by the Authorities, who are directed by the Agricultural Department. The same disinfecting measures were at once taken. The anthrax having broken out at Gardens Farm, Mr Bell suspected the linseed cake. Mr Bowlas had sent for Mr Bell. On the second occasion both Mr Bowlas and Mr Bell were present. In consequence of a direction given by Mr Bell, the linseed cake was entirely stopped on the 28th. Two other bulls died on the 29th and 30th March at Eamont Bridge Farm. They were buried in the same way. There was no further outbreak of anthrax. The last death was on the 30th March. Lord Brougham then called on Professor Williams and Professor M'Fadyean to make an inquiry and report. The square feed cake and linseed cake were bought from Messrs Pattinsons & Winter of Penrith, the Defendants. As regards linseed cake, there were 6 cwt. on the 12th March, 18 cwt. on the 12th, another 18 cwt. on the 12th, 6 cwt. on the 21st, and 16 cwt. on the 28th. That was all purchased and paid for. After its use had been discontinued some of it was left over. The cake was marked in a special way, so as to be easily identified, and certain portions of it were handed over to Messrs Armstrong, the Solicitors. The cake which had been bought for a considerable number of years was "97 per cent pure No. 1 linseed cake." He believed that some of the cake had been used. Sometime in 1891, a bull died at Eamont Bridge. A veterinary surgeon, Mr Armstrong, was called in. He had the body opened and was of opinion that it was anthrax. On the 10th of April, the bull was buried. Between 1891 and 1899, there was no case of anthrax at the farm. Up to March 1900, the cattle were in a healthy condition.

Brougham has a balance sheet made up once a year. It is made up for him, and a valuation is made by some farmers in the neighbourhood so that he may see how he stands. The surplus stock is sold from time to time, chiefly to go abroad. Lord Brougham does not send his stock into the market to be sold, but from time to time they are sold, as being valuable stock. They have been sold from 30 guineas up to 300 guineas. The value given in the books of the animals lost through anthrax was £225.

Cross-examined by Mr Pickford.—Mr Westgarth said that Messrs Pattinsons & Winter supplied not only Lord Brougham, but a great many other farms in the neighbourhood. Very likely the same kind of cake was supplied. He thought that some of Lord Lonsdale's farms were supplied, and a good many others. He had heard of no outbreak of anthrax on any of these farms. On 5th March, 6 cwt. of E. K. linseed cake was supplied. On 26th February feed cake only had been supplied. On 12th March, 18 cwt. was supplied to Hospital Farm and 18 cwt. to Eamont Bridge. On 21st, 6 cwt. went to Gardens Farm. There was no outbreak at all at Hospital Farm. The 6 cwt. supplied on the 6th March would be finished before the 12th. A fresh supply was received on 12th March. One lot is finished before another is supplied. The date on which he received the samples which he sent to Professor M'Fadyean would be the 12th. He sent them to Professor M'Fadyean on 2nd April. On 28th March, 16 cwt. E. K. went to Hospital Farm. The samples which he sent were taken from Eamont Bridge Farm. He took them, put them together, and sent them himself. He sent about 2 cwt. Professor M'Fadyean came down on the evening of 30th March, and stayed until the 31st. The samples were sent to him on 2nd April in a sack. The cake had been kept where cake was always kept, in the store-room. He sent samples of the supply received on the 12th. There are different men on the different farms, and every man attends to his own lot. There is no foreman or head man who attends to all of them. He (Mr Westgarth) goes from one place to the other, and nobody else. All the others are separate staffs, so to say. On the 9th, he saw both Mr Pears and Mr Bowlas. One of the bulls had been skinned and partly dressed before they arrived. This was very likely done to find out what ailed the bull. It was not skinned and dressed as an animal would be for a butcher. The whole of the skin was off. They only wanted to find what had caused death. The entrails had been taken out, just opposite the place where it was buried. It had died in the box, 50 or 60 yards from where it was buried. It was taken from the box to the burial place in a float. The second bull that died on the 9th was not skinned or dressed at all. That animal died in the large cattle court, which was shown in the plan. That was buried in the same place, and removed in the same way, in a float. Two of their own men did the skinning and dressing. These men had been attending on the cattle, and continued to do so afterwards. He now knew that if an animal died of anthrax, it was most important that the carcass should not be opened. He did not know that at the time. The float was backed up to the door, and then the carcass was dragged along the floor into the float, and taken to the place where it was buried. It was after getting Professor M'Fadyean's report that he communicated with Messrs Arnison & Co., Lord Brougham's solicitors. He knew that they communicated with Messrs Pattinsons & Winter. He did not know that they in turn communicated with the makers of the cake. He did not see the letters written by Messrs Arnison & Co. He knew that they, acting for Lord Brougham, declined for a long time to allow either Messrs Pattinsons & Winter or the makers of the cake to visit the premises or make any inquiry at all. On 15th May the makers made an examination of the ground where the outbreak occurred, and drew samples for examination. He believed this had at first been refused, the application having been made towards the end

of April. He did not know whether the reason given for refusing was that some other theory might be started as to the cause of the disaster. None of the cattle ever went into the paddock where the 1891 animal was buried. This paddock was railed off simply as a place for implements—a sort of rubbish place. Cattle were never put into it. The men were about there. He was there in 1891. The animal which died in that year was also skinned and dressed, or partly dressed, the entrails removed, and so on. This was done in the same place—the paddock. It died in one of the boxes in the large cattle court. It was taken to burial on a sledge, the carcase being kept off the ground altogether. Messrs Earle & King, the makers of the cake, came on the 25th, and took samples away. He believed that Mr Winter had said something about taking away the bulk of the cake to feed cattle with. He said he would take it and run the risk. He did not want to take it particularly. He said, "I will allow the price and take the cake, and feed my bullocks with it." A gentleman with Mr Winter took samples. The defendants were allowed to have a portion of the sample that Professor M'Fadyean had taken, in order that they might test it. This was a portion of the same sort of cake that had been sent to Professor M'Fadyean. They did not ask for a portion of the same sample that had been sent to Professor M'Fadyean. To his knowledge all the other things the cattle were fed upon had not been analysed. Professor Williams took them all; hay, straw, turnips, meals, bran, Indian meal, and everything. He did not know whether they were analysed. The bran was supplied from a miller's close by. It had been supplied by Pattinsons & Winter as well, until shortly before this time. It was supplied from these two sources alone during February and March. He could not say whether any had been supplied by Pattinsons & Winter all those months until the cake was stopped. He did not know where the bran obtained from the miller came from.

Re-examined by Mr Jelf,—the witness said he knew Mr Riley. He thought Mr Riley's farm was about five miles west of Penrith. Mr Riley's man had told him (Mr Westgarth) that they had lost a bullock from anthrax, which had been fed with the same cake. The cake had been sent by Pattinsons & Winter. He saw no notice given about that. Lord Brougham had a regular store place for the stuff. It is properly attended to and kept in order. He did not think there was anything there to infect the cake. The lot which came on 5th March was all consumed before his attention was turned to that as being likely to have caused the mischief. The first bull that died in 1900 was in an outside box, in the yard near the tennis court. The second bull died in the cattle court, at the other end. That was some distance from the place where the one died in 1891. The cow died in the byre in the yard; a separate building altogether. There is a gate into the place where the bull died in 1891. When the bull died in 1891 the usual precautions were taken, under the Authorities, about the burial. Mr Armstrong (the veterinary surgeon who was to be called on the other side of the case) was present. Professor Williams took away some of the things to analyse. After the anthrax had been discovered and the linseed cake stopped the animals were fed with the same foods as before. Nothing at all happened in the way of illness. Lord Brougham did not keep a stock book; the deaths were recorded in a diary, with the cause of death as far as it was known. All the animals which died from 1891 to 1900 had the cause of death recorded. He had made out a list from his diary. A veterinary surgeon was called in on each occasion to find out the cause of death.

The witness recalled,—said that the hay and other things which had been given to these cattle were also given to other animals on the farm. The horses had hay and corn. The sheep had oats and turnips and that sort of

thing. Part of the food stuff used to be given to them, and no harm came to them.

Mr THOMAS BOWLAS, *examined by Mr Jelf*, said that he was a Member of the Royal College of Veterinary Surgeons, carrying on business at Penrith. On 9th March 1900 he attended at Lord Brougham's Eamont Bridge Farm and examined two bulls which had died. On examination he found that the cause of death was anthrax. The Local Authority then took charge of the carcasses and buried them in the usual way. Anthrax is a disease produced by introduction into the internal organs of the animal by the food, or it may get into a wound. It may be either in the throat or stomach. On his examination of the carcasses he observed signs of anthrax, the spleen being much enlarged and the bowels containing blood. He had had considerable experience of that and other diseases. Every precaution was taken in the way of burying the carcasses and preventing mischief happening. The place where any blood of the animal was lying was disinfected with a strong solution of carbolic acid—crude commercial carbolic. The box was white-washed, and the men who had anything to do with the animal were ordered to immediately change their clothes, to disinfect their boots, and to wash themselves thoroughly with an antiseptic. He at first thought it was the square cake, and ordered it to be discontinued. The linseed cake was substituted for the square cake at the Gardens Farm, and a three-year-old cow died there on the 27th. He examined the cow after death, and found death due to anthrax. Two days previously a cow had died at the Eamont Bridge Farm, also from anthrax. He believed he had seen this cow when alive before it was ill. The symptoms were such as showed anthrax, or the probability of anthrax. When he examined the carcasses he came to the conclusion that it was anthrax. He believed that there had been no other feeding stuff besides the linseed cake in common use between Eamont Bridge and Gardens Farm. On the 28th March, in consultation with Mr John Bell, veterinary surgeon, Carlisle, the use of the linseed cake was stopped. On the same day a bull was taken ill, and died on the 29th. From his knowledge of the disease that was a thing which might have happened although the linseed cake was stopped on the 28th, the illness coming on from what the animal had been eating before. The usual time of incubation of a disease of this kind is from two to seven days. The last bull died on the 30th, also from anthrax. On the 29th he met Principal Williams of Edinburgh, who inspected the Eamont Bridge and Gardens Farm and the feeding stuffs. Principal Williams saw the last dead bull and took away a bit of the ear. He (Mr Bowlas) met Professor M'Fadyean on the evening of the 30th March, and was with him on the 31st when he examined the buildings on the two farms. None of the carcasses were dressed for sale, but one had been opened. Every precaution was taken to prevent any danger from the carcasses, all the natural openings being stuffed with tow and strong carbolic acid. The carcasses were taken to the burying place in a float, and care taken that no blood or anything else could fall from the carcasses.

Cross-examined by Mr Pickford.—Mr Bowlas said that when he first saw them one of the carcasses was in the burying paddock, and the other was where it died. He was referring to the first two, on the 9th March. The one in the paddock had not been dressed, but skinned and opened, and the entrails taken out. This carcase had been removed before he came. He understood that the natural openings—nostrils, and so on—of that carcase had been plugged before it was moved. The other one he saw done. It was done to prevent any blood or anything of that kind escaping. Every drop of blood in an animal which dies of anthrax swarms with these bacilli. It is a pretty well accepted thing that if an animal is suspected to have died of anthrax it had much better not be either skinned or opened or anything

of the kind done to it. Certainly it should not be done by people who are going amongst the cattle afterwards. To some extent it is difficult to tell when an anthrax bacillus is got rid of when it once gets to a place. It was for their own benefit that he told the men to disinfect themselves, their clothes, their boots, and everything else. If the bacillus gets into the blood it attacks human beings. Unless the disinfection and cleansing was of the most complete nature, for men to handle a carcase which had been opened and then to go amongst the other cattle and amongst their feed would be a very likely way to convey the bacillus; but in this case he believed that every possible precaution had been taken under the circumstances. The men who had anything to do with the carcasses were not allowed to go to the other cattle, but sent straight home to change. Unless disinfection is thorough, for people who have handled a carcase of this kind to go amongst the other cattle and the food is very likely to convey the bacilli. One animal on the 9th was skinned before he arrived. He could not say how long it had been skinned. The skinning and opening operation was finished when he arrived. As far as he could understand, the men had got so far with the business to find out the cause of disease, and became suspicious and sent in. Only one man was in the paddock where the beast was when he arrived. The anthrax bacillus is a very deadly thing to cattle, but it does not follow that every animal which takes the bacillus gets the disease. At the same time, cattle are very liable to it. He did not think that younger cattle were more susceptible as a rule than the older. He had watched anthrax as a veterinary surgeon in various cases. He thought that younger animals were not more susceptible, and that any animal is liable to take anthrax though young. The young are just as susceptible as the old. He practised as largely as he could in this district, and he knew that this E. & K. linseed cake was a good deal used in this district. He understood that it was used on several of Lord Lonsdale's farms and a number of others in the neighbourhood. About this time he had no cases of anthrax arising on any of these other farms under his special notice, nor did he know of any in the district. He knew that there had been a case in 1891, and Mr Jelf had referred to one somewhere later on. He knew that the linseed cake had been substituted at the Hospital Farm. He could not say with any certainty whether some was sent to the Hospital Farm on the 12th March. He thought it was a few days after the first two bulls died. He did not know what quantity of cake had been used. There were no deaths from anthrax or any other cause that he knew of at the Hospital Farm. There was only one death from anthrax at the Gardens Farm. The linseed cake had been used at the Gardens Farm a few days before the animal's death. He did not know how many animals were at the farm, but the number was considerable. There were about forty at Eamont Bridge, about nineteen at the Gardens Farm, and about forty-eight at the Hospital Farm. Eamont Bridge had considerably the worst of the outbreak. To his knowledge there had been a previous case of anthrax at the Eamont Bridge Farm. The anthrax bacillus produces what are called spores. Under certain conditions they remain dormant for years, and yet preserve their vital power, while under others they die. He was not a bacteriologist, but he understood that if the condition in which these spores are is not suitable to them, then they die. They must have a suitable place to live in. He did not know exactly under what conditions the spores would preserve vitality for a number of years. He believed there were some such conditions. They will preserve vitality for a great number of years, lying dormant in the meantime. He could not himself test the cakes or the feed or anything. He opened one animal, but did not make a *post-mortem* of the other animals. He contented himself with a microscopical examination of the blood, a little of which he took on a slide. The others were not opened

at all. He had been attending these farms of Lord Brougham's since 1898. He believed that he or his partner had been called in to all the animals that had died since then. He believed his partner attended the first case in January 1898, which died of muco-enteritis. As far as he remembered he did not himself attend. The next died from serofulous liver, the next milk fever, then inflammation of the lungs, and then blood poisoning following damaged eye. He attended some, but not all, of these. He never attended at Lord Brougham's farm until after September 1898. He believed that was the first animal of Lord Brougham's that he attended. He did not remember the case of a red bullock on 1st June 1899, which was described as "After four days' illness."

Re-examined by Mr Jelf,—Mr Bowlas said that he believed it would be about half past four when he arrived at the farm on 9th March. He judged that the animal, which he found skinned and partly dressed, had been dead a few hours, but could not say exactly. The weather was frosty, which would militate against the spreading of contamination. As far as he saw, there did not seem to be anything that had happened in the way of the coming away of matter or anything of that kind from the animal. Infection might be carried by the germs being carried about, or lifted in the dust, or anything of that kind. It does no harm unless it gets into the blood. Since 1898 it had been the practice of himself or his partner to go down in case there was any animal which had died or was ill. They then gave the statement of what they judged to be the cause of death to Mr Westgarth. He understood that this was written down in a diary, but had not seen it done. From 1891, when the bull died, other diseases were given for every one, such as—"Tuberculosis, red and white heifer," "blood poisoning following damaged eye." That would have nothing at all to do with anthrax. He did not recollect "Red bullock, after four days' illness, on 1st June 1899." In the regular course, he or his partner, if a death occurred from anthrax, had given that cause of death to Mr Westgarth. There are unmistakable signs where there is a case of anthrax. It was not a thing either of them would be likely to mistake.

MR JOHN WESTGARTH, *recalled and further examined by Mr Jelf*, said that the bull which had been skinned died at 1 P.M. It was skinned and partly dressed immediately afterwards. Two persons took part in the operation. Every precaution was taken in the way they did the work. Skinning was done in the ordinary way. After skinning the men washed themselves, changed their clothes, disinfected their boots, and everything else of that kind. That was before the veterinary surgeon arrived. He (Mr Westgarth) suspected the case to be one of anthrax. The men did not go near any other cattle or cattle-place until after they had been disinfected. The same precautions were afterwards taken with regard to each animal that died.

Further cross-examined by Mr Pickford,—Mr Westgarth said that before the veterinary surgeon came he told the men to wash themselves, change their clothes, and disinfect their boots with carbolic. He suspected it to be a case of anthrax, but had not done so when he ordered the animal to be skinned and opened. At the time when the animal was opened he did not know that it was a very dangerous thing to do, presuming it to be a case of anthrax. He did not know that skinning and opening the carcase was a very likely way to spread it, if it were anthrax, or he would not have had it done. He was there all the time, from giving the men their orders until the veterinary surgeon came. He could not say that he had his eye on the men all the time during those two or three hours. The men were sent to change their clothes; one had come back when the veterinary surgeon came; very likely the other had gone to do some work, he did not know where. These men had been in Lord Brougham's employment under him for some time.

MR JOHN JACKSON BELL, *examined by Mr Sturman*, said that he was a

member of the College of Veterinary Surgeons, and the Chief Inspector for the County of Cumberland, and also practised on his own account. On 28th March 1900 he was called in by Mr Bowlas, to go to Lord Brougham's farm at Eamont Bridge to consult with him about this outbreak of anthrax. He made all inquiries as to the food and treatment of the cattle. He looked at the sheds and buildings; they were good, clean, and well kept. He made inquiries as to all the food with which the different animals had been supplied. It was his opinion that no more of the linseed cake should be supplied. Anthrax is a disease which must be either taken in food or water. It was his usual custom to discover the cause of an outbreak by inquiring about the different foods and eliminating those which could not have caused it. As County Authority and privately he had had a great deal of experience with regard to outbreaks of anthrax. It could also be communicated by inoculation. In his opinion it might possibly get into the mouth, if floating about in the air, and taken in from the air. But his opinion was that it was ordinarily communicated through food and drink. For twenty-five years he had been dealing with anthrax. For a long time they were not aware of the germ existing, although the disease had existed for a greater number of years. He had known the disease, but was not acquainted with the specific cause of it. He had been Chief Veterinary Inspector to the County of Cumberland for close upon forty years. He saw several of the animals, but he saw one, a young bull, that was then suffering from the effects of anthrax. This bull was attacked with anthrax; he noticed it to be ill on the 28th; it died on the 29th. This was the only animal that he saw affected with the disease. He had no doubt whatever that it was suffering from anthrax, and stated that he thought it would be dead next day, which was verified. He recommended that the linseed cake should be discontinued. He gave it as his opinion that if this was done the disease would probably cease. He had generally found the period of incubation of anthrax to be from two to seven days. Assuming that a cake like this could be infected, it was quite reasonable to expect that the bacillus might be found in one part of the cake and not in another. It was possible or probable that an animal taking a tainted cake, might not catch the disease. The reason was that some animals are more susceptible to these attacks than others. If the food containing this passed through without there being any lesion, or without its getting into the blood, it would be harmless to the animal, and would form part of its excreta. There was an outbreak at Mr Riley's, the date of which he could not give, but it was a considerable time back. Some time ago he attended Mr Riley's. It was probably within two or three years; he could not give the exact time. He was called in by the County Authority in consultation. The cattle that died had been fed upon some cake, whether linseed cake or what cake it was he could not state. He formed the opinion that the cake was the cause of the outbreak, and asked for its discontinuance. From close observation he had noticed that most of the cases that he had been called in to examine had arisen, in his mind, from feeding stuffs, and largely cakes.

Cross-examined by Mr Pickford.—Mr. Bell said he did not know what particular cake had been used for Riley's cattle. He ordered its discontinuance, but unfortunately after a cessation of giving that food for two or three weeks, the cake which had been stored away with the intention of not being used again was got at by a cowman, who gave it to some of the cows and killed a cow. There was a second outbreak, and after that there was no more disease there for a time, at least two years. There had been an outbreak a very few months ago at this farm, since the beginning of this year. If the cake which had been substituted at Riley's after the former outbreak were the E. & K. cake, that would be a point in favour of this cake. He always tried to find out if possible what was the

food most likely to have produced the disease. He always recommended a change of food if he suspected that it arose in that way. He did not know whether the cake used at Riley's two or three years ago was an undecorticated cake, made in Glasgow. He did not know that they substituted the linseed cake after that. In order to produce anthrax the bacillus must get into the blood. The germs or spores adhere to the food. If taken in food, it passes into the blood through the intestines, sometimes through a lesion in the intestine, and sometimes without. It may be floating about in the air. He understood that human beings ordinarily took it by inhaling, in what is called woolsorters' disease. This is taken by inhaling the bacillus which comes from the wool. It may also be taken by being introduced into an external sore or cut. He agreed that under favourable circumstances if it produces spores they may remain dormant for years. The spores must be carried, as they have no power of motion. They may remain dormant for a good many years, and they may be carried and become active. They may be carried upon the hands, or clothes, or boots, or anything else. They may be carried by animals, cats, dogs, rats, flies, anything. If they get upon the animals, of course they can carry them from one point to another. A spore may be carried by getting upon the boots or clothes of men, or upon the skins of animals, of cats or dogs, or anything of that kind. The spore is given off from the bacillus, and becomes divided. The spore is not visible to the naked eye. The spore is a very small particle indeed of the bacillus, and therefore much smaller. He had read of cases in which the spore remained dormant for many years, and afterwards became active. If there is an outbreak of anthrax, there may be spores existing which may become active, and produce another outbreak years afterwards.

Re-examined by Mr Jelf.—the witness said that it was possible, but in his judgment very improbable, that if a bull died of anthrax in April 1891, and was immediately buried with all precautions, that nine years later there would come up a case of anthrax in that farm, having arisen from certain spores dormant during the nine years. The outbreak at Riley's was stopped by discontinuing the cake which produced the disease. It was apparent that if that so-called harmless cake was being given, and was going on in January 1901, that would be at the same time that this fresh outbreak of anthrax happened at Mr Riley's. He was not called in respecting this case; he was consulted. He knew all about it, but did not consider it necessary to go over, because the animal was buried when he got notice about it.

Professor JOHN McFADYEN, *examined by Mr Jelf*.—said that he was the Principal of the Royal Veterinary College in London. On the 30th March 1900 he went to Brougham and inspected Lord Brougham's herd of short-horn cattle. He did not see any of the dead animals, as they were buried before he arrived. He examined the building and made inquiries about the food and so forth, his business being to discover the cause of the outbreak. He attributed the damage to the linseed cake, and determined to examine it. It appeared to him that the circumstantial evidence excluded every other class of food, and also the drinking water. He suspected the linseed cake, and this theory fitted in absolutely with all the circumstances. He received some morsels of the cake on the 31st or 1st. He received two separate parcels. He received a quantity of pure linseed cake in a bag, and he received two small cakes, one of the linseed cake and one of what has been called the soya-bean cake, together. He secured for immediate experiment the whole of the linseed cake which had been sent. He resolved to test it by what was supposed to be the most delicate method of discovering anthrax germs in food materials. He prepared a number of small portions from different morsels of the cake, and put each morsel into a test-tube, and added a sort of muddy

liquid, and strained this through muslin, and then he injected about eighteen or twenty drops of the muddy liquid into each of two guinea pigs, and fifteen times that quantity into a sheep. That was on the 7th, not on the 6th. The usual precautions taken in such experiments were observed, all instruments used being submitted to boiling temperature. The water was boiled also. That was sufficient to kill all anthrax germs, even in the state of spores. On the morning of the 11th he examined the experimental animals, and it was found that one of the guinea pigs was moribund. He had it killed immediately and submitted to examination. He found that it had, at the place at which the liquid had been injected, the alteration which is almost characteristic of anthrax. On opening it he found it had enlargement of the spleen, which is also fairly characteristic of anthrax. He then proceeded to make a microscopical examination of the liquid taken from the point at which the infective material had been put under the skin, and also to examine the blood. In both cases he found that the liquid was swarming with anthrax bacilli. He also proceeded to obtain the bacillus in a state of artificial culture from the guinea pig, inoculating artificial medium in a tube with a little of the pulp from the spleen, and put this to incubate, and this yielded a perfectly characteristic pure culture of the anthrax germ. The other guinea pig and the sheep survived, and showed no signs of disease. This quite confirmed the opinion at which he had arrived on the circumstantial evidence alone; that the cake actually infected the animals that died at Brougham. The fact that the other guinea pig and sheep escaped did not militate at all against that. The quantity of cake actually used in his experiment would probably be less than a grain. It was scarcely possible to conceive of any natural circumstances that would contaminate a quantity of food material throughout every part of it. There was an analogy in the case of his two guinea pigs and the sheep, and what happened at the farm where the other cattle escaped. He had omitted in connection with his experiments to say that he had made a microscopic preparation from the spleen of the guinea pig, and that he had also a photograph of the artificial culture obtained; he had also with him a tube of the anthrax bacillus which was isolated. The small rod like things in the photograph to the right were the bacilli, and to the left were the same bacilli which had grown out much longer in the artificial culture. The spores were the small specks in the photograph. He knew where linseed came from, and what process it goes through. On a former occasion he had had to go into the whole question of the possibility of linseed contamination from anthrax, and he followed the process of preparation in one large factory, where he supposed the usual method was adopted. He believed that linseed was mostly obtained from countries in which anthrax is a much more prevalent disease than in this country, and where the precautions taken by the authorities are sometimes absolutely nil. It was quite conceivable that, in these places, there might be rather extensive contamination of linseed. He might say that almost every European country has a great deal more anthrax than Great Britain, and particularly those in which there is a hot summer temperature. In Russia, for example, and in Turkey, anthrax is very prevalent. He did not know where the cake came from abroad. Any agency which would transfer the germ from one animal to another would be liable to object that open, but, on the other hand, without exception, authorities were agreed that the most common mode of infection in cattle was by the germ in drinking water. On the question of the cake, the question whether the cake could be infected by the germ was not settled, but it was not impossible that anthrax if they had got some of the germ from the cake.

appear to him to be at all a reasonable explanation of the facts; it would have to be utterly rejected as an explanation of the death of the cow at a farm half a mile off. In his judgment it was not at all satisfactory as an explanation. He had no doubt that the particular cake which he examined was infected with anthrax. If it could be established that there had been any transference of blood from one of these animals to the cake, of course that would infect the cake. Only gross carelessness could produce contamination. There was an immense experience to prove that such precautions as had been sworn to having been observed were usually efficacious. The average number of animals infected in outbreaks so dealt with throughout the year was less than two. With regard to the case of the 1891 death, assuming the facts to be that in April 1891, in the inner box which was shown on a plan, some little way off from where this animal died, a bull died of anthrax, and was immediately buried with the proper precautions, in his opinion it was utterly unreasonable to suppose that that could have anything to do with this outbreak of nine years afterwards. Anthrax is not an infectious disease in the ordinary sense, and as he had just stated, in the hundreds of outbreaks which occur during the year, the average number of animals affected is less than two. As a rule, where anthrax is diagnosed amongst cattle and a complete change is made in the feeding materials, and thorough disinfection and burial of the carcase is practised, the disease stops. On, he thought, the 27th November, he forwarded to Professor Delepine a culture of the anthrax bacillus which was obtained from the guinea pig of his experiment, exactly the same as was contained in the tube which he had produced, and also two microscopic preparations similar to the photographs.

Cross-examined by Mr Pickford,—the witness said that he had suggested that the bacillus got into the cake by adhering to the linseed before manufacture. A boiling temperature, in a moist state, maintained for two or three minutes would destroy the spore. He found it impossible to believe that in the preparation of this cake the whole of the linseed was subjected to a heat of about 280 degrees, because the cake swarmed with bacteria of other sorts throughout its whole substance, and these also would have been destroyed by a boiling temperature maintained for two or three minutes. That statement he based on his own bacteriological examinations. Assuming that the bacillus was killed in the process of manufacture, then it must have got into the cake subsequently. The bacillus would have been destroyed if subjected to the afore-mentioned heat. If the bacillus were adhering to the linseed, he should expect it to adhere to the particular seeds for the most part with which the infective material was originally brought in contact. No doubt there might be a partial infection of other seeds in proportion to the extent to which the linseed was mixed, but he did not think it was likely the infection would be uniform throughout any large mass. He would not expect it to be in every grain of matter. Supposing a consignment of linseed to be infected in the way he said, and a large quantity of cake to be made out of it, he would not expect the infection to be pretty well spread over that make of cake. He did not think it likely that any large consignment was anything like infected throughout and throughout. His impression was that the contamination would always be rather localised than general. It seemed to him perfectly likely that only half a ton of linseed might be infected. If all that went to one farm one could understand it then being confined to the cake there. If the half ton of infected cake were distributed to different farms, he should expect disease at each farm, but he meant that it would not be improbable that half a ton of infected cake might be sent to one farm. He knew Professor Boyce personally, and also Professor Klein. He knew Dr

Sims Woodhead intimately. He also knew Professor Muir. He thought all these gentlemen would be likely to recognise the anthrax bacillus if they saw it. Supposing that these gentlemen had had samples of the same consignment of cake from which his had been taken, and had found no anthrax bacillus, he should not think that the anthrax bacillus was not there. Unless the examination were very prolonged—so prolonged as to be practically impossible—no examiner would be justified in saying on the negative result of an examination that anthrax spores were not there. He was not prepared to assent that the negative result of an examination would justify the statement that the spores were not there, even in the particle analysed, as he was not sure that one spore in twenty drops of water would infect. He thought probably it would. This was the first time he had ever found the bacillus in linseed cake. He had had a number of cases in which the circumstantial evidence had pointed to it, but he had not been able to find the bacillus on experimenting. Supposing cake made from the same consignment of linseed to have been supplied to a great number of places, and no ill effects from it, and also samples of this cake to have been examined by the gentlemen mentioned, and no bacilli found, that would point to local contamination, in the sense that it was sent only to this farm. Opening or skinning the carcase of an animal that has died from anthrax is always dangerous to the person who conducts the operation. When very great precautions are taken, the risk, when apprehended, of the bacillus being carried by such a person, is not great. The ordinary measures directed by the Board of Agriculture were so efficacious that, as a rule, there was no second case. Supposing a man had been operating on an animal by opening or skinning it, and washed his hands afterwards, he might, provided he immediately handled food materials, to be given at once to another animal, convey the bacillus to the food materials; it was highly improbable, but possible. It would entirely depend upon the feeding material whether the bacillus in it would multiply or not. In ordinary food materials it would not multiply, and would probably die. Cake does not contain the moisture necessary for the bacillus to multiply. It would only be conveyed to the outside of the cake, and in all probability would very soon be dead, but if given to animals soon it might convey the bacillus. The bacilli are much less resistant than the spore. There had been no spores formed in connection with the Brougham outbreak; circumstances had not made it possible for spores to be formed in that outbreak during the month of March. No sporulation takes place below 60° Fah. There was no such temperature in the month of March. He also ventured to affirm that no spores were formed in connection with the animal which died in April 1891. To produce spores a temperature of about 64° would be required for a week. He was expressing his positive opinion, but he admitted that that was not necessarily a positive fact. The samples came to him by rail. The temperature inside the cow-house might be 60°, but considerably more than 60° would be required. He had stated that sporulation did not take place below 60°. He could not say where the samples came from; all his communications had been with Messrs Arnison, the solicitors, who told him the samples were coming. They came in two separate parcels; one in a sack, the other wrapped in paper. One drop of blood from an animal with anthrax swarms with anthrax bacilli. If one drop of blood had been conveyed in any way to the food, it might contain a great number of bacilli. The skinning and dressing of an animal means the liberation of countless numbers of germs. There is a possibility that they may infect the hands of those who conduct the operation, or give rise to further cases of anthrax in animals by being taken in food and water.

Practically the only way in which cattle are infected is by the bacillus being left on the exterior of food materials, or introduced into water. Any mechanical agency that would transfer the blood or discharges from the carcase to any solid object would contaminate that object. He powdered the sample of cake sent to him. He broke it up with a heated instrument and selected the inside particles as far as possible. He did not break it all up: he simply took the chips. He could not say that he took off the outside and powdered the inside only. He broke it up so as to get a number of pieces of a size convenient for trituration in a mortar, and selected those pieces as far as possible from the inside. Where extensive outbreaks of anthrax occur, he thought they were very often the result of carelessness with the first case; but the causation of anthrax occurring in the fields is often entirely obscure. It is almost the rule that it is not possible to put one's finger on the cause of the outbreak. When an animal is in the field it is not possible to ascertain the exact spot of contamination. Although the average number of animals attacked by outbreaks in this country is less than two, in occasional cases the number is very much higher, and with rare exceptions these more serious outbreaks are the result of ignorance or carelessness in dealing with the first case. This case was one of the rare exceptions. He agreed with the following quotation: "The cause of anthrax is a microscopic germ, the anthrax bacillus which multiplies in the bodies of infected animals, chiefly in the blood stream. An animal that is the subject of anthrax is to some extent dangerous to its fellows in the same field or building while it is still alive, because of the germs which are apt to be passed out with the excrement or urine, but it may become an infinitely greater source of danger after death, if, without any precautions in the way of disinfection the carcase is skinned or dressed." He really did not think there had been any risk involved in the way in which the sample had been sent to him in a sack. He would not have objected to taking it that way himself, as long as the thing was taken in circumstances that made it unreasonable to suppose that it had been contaminated accidentally. He saw the bag in which it came; he did not remember whether it was old or new. His directions had been that samples were to be sent to him from the lot of cake which the animals had been eating at the time of death. He thought it would be utterly unreasonable to suggest that an animal being skinned and opened might have carried the infection to two or three different places, one of them half a mile away. It was more difficult to accept such a suggestion when the succeeding cases occurred in two different buildings. He had found that the same food had not been given to animals in different buildings at Gardens Farm, but at Eamont Bridge they were fed by the same men and had the same food. It would be an extraordinarily difficult thing to explain why only the linseed cake was infected, if the cases at Eamont Bridge were due to the fact that the infection had been carried to the food. If the circumstances were such as to carry the contamination to the linseed cake only, leaving the oats, bran, hay and straw, and everything else uncontaminated, one might expect, as an outside possibility, that it did not matter what houses the animals were in. The other foods were not analysed, but were subjected to a searching test, inasmuch as they were continued for food for months afterwards. He thought that these other food stuffs did occasionally contain the anthrax bacillus. He thought that there was no class of food stuffs that could be excluded in that way.

Re-examined by Mr Jelf.—The witness said that the following passage was taken from the *Journal of Comparative Pathology and Therapeutics*, which he edited, and was contained in his annual report for 1900 to the Royal Agricultural Society: "During the past year several samples of cake and oats were sent to the laboratory to be tested, in consequence of suspicion that the

material in question had caused anthrax among animals fed thereon. Although in all these cases it was recorded that the circumstantial evidence pointed very strongly to the presence of anthrax germs on the cake or oats, the experimental tests gave negative results in all but one case. In that instance it was ascertained that anthrax germs were undoubtedly present. That was the cake, and in his annual report he had given that as his scientific opinion and published it to the world. He believed that to be correct.

Professor SHERIDAN DELEPINE, *examined by Mr. Stearns*, said that he was the Professor of Pathology at the Victoria University and Director of the Pathological Laboratories at Owens College. He was Professor of Public Health at the Victoria University, and was bacteriologist to fifty or sixty public bodies. On 19th November he was brought in to give his assistance in this case. Professor M'Fadyean sent him three specimens—one tube of culture of bacillus anthracis similar to the one which Professor M'Fadyean had shown, and two microscopic preparations. The culture of anthrax was in a tube; this he examined microscopically and also by inoculation. The bacilli in the culture were typical anthrax bacilli. He inoculated two guinea-pigs with minute quantities of the culture, taking every possible precaution to avoid infection of the wound at the same time, or accidental contamination of any kind, and within thirty hours both animals died, and showed the most typical lesions of anthrax. They were literally swarming with the anthrax bacilli. He had not the slightest doubt as to their being anthrax bacilli. He made some cultures from the original tube in bouillon and agar. All the different cultures gave typical growths of anthrax. He made two tests and examined these bacilli microscopically, and found exactly what he expected for anthrax bacilli. He examined the microscopic preparation from the spleen, labelled "Spleen, guinea-pig. Brougham, April 11th." This was a portion of the preparation of one of the original animals inoculated by Professor M'Fadyean. He found that these preparations showed a large number of absolutely typical anthrax bacilli. He corroborated what Professor M'Fadyean said, that these were undoubtedly anthrax bacilli. The third preparation, marked "Second culture from Brougham guinea-pig," was apparently a preparation made from a culture also made by Professor M'Fadyean, he thought what they called a sub-culture: the second or fourth; he did not remember; and this preparation was evidently one of anthrax bacillus—absolutely typical. He had heard the statement that there had been an outbreak at this farm in 1891. With regard to the theory that the outbreak nine years afterwards was due in any way to the earlier outbreak of 1891, in the absence of any case during the interval, he thought such a theory very far fetched. He did not know what to say as to the suggestion that the sample of cake sent to Professor M'Fadyean could have become inoculated with the anthrax bacillus after it had reached the farm. He had not visited the farm, but from the plans which he had seen, the situation of the cake house, the situation of the place where the animal was skinned, the precautions which were taken afterwards to cause disinfection of the soil, and also disinfection of the hands, boots, clothes, and the sending away of the men—all these things seemed to him to show that the possibility of contamination of the cake in the cake-house, which was so far off, was most improbable. He got a sample of the cake from Messrs Arnison, and in January, he thought about the 26th, of this year he sent one of his servants to Liverpool to obtain a good sample of the E. & K. cake by applying to Messrs Earle & King. He got this cake on the 26th or 28th of January. He had some samples of the original cake and some recent samples to compare them together to see whether any change had taken place in the process of manufacture. This year, for another purpose, he procured two samples, and one sample was given to him by Messrs Arnison, all with E. & K. on them. He took some

parts from the central parts of the cake as carefully as possible, avoiding any surface parts. He examined them bacteriologically, and found that they were teeming with spores of bacteria, but he found no anthrax. He afterwards subjected portions of the same cake to a temperature of 100° C. After half an hour's steaming at 100° C., that is 212° F., the cake was entirely sterilised, and after another fortnight he found that it had remained absolutely sterile. All the other bacteria he found there were killed after being exposed to 212° F. When he found them in all the three samples they were alive. The bacteria which were present in the cakes he examined were not more resistant than the anthrax bacillus. The total time of exposure to a current of steam at 100° C. was not more than a quarter of an hour, and considering the nature of the material, which was porous and difficult to penetrate, he thought anthrax bacilli would have behaved very much in the same way as the spores which he had found present in that cake. This experiment showed that if the same heat of 212° F. had been applied to that cake, the bacilli which he had found in the cake could not have been living. It was not only a question of temperature, it was a question of steam. If steam free from air and well distributed through the mass had been allowed to pass through the cake, it would have been entirely sterilised. This experiment proved that, in the three samples of cake he had had, there had not been sufficient heat supplied to sterilise the bacteria in the cake. The bacteria he found were taken entirely from the interior of the cake. In fact he seared the surface of the broken cake with a hot iron, to remove any possibility of the dust from the surface getting in, and he took the central portion at least a quarter of an inch from the surface.

Cross-examined by Mr Pickford,—the witness said that he did this to ensure that there should not be any of the common bacteria usually found in dust in his experiment. If it is required to test whether it is in the cake, the right thing to do is to sterilise the outside. If one of the bacilli with which he had said the preparations were swarming were put into a guinea-pig it would very soon reproduce itself in the guinea-pig. The exact number of bacilli necessary to infect was not known, but it might be admitted that at least two, three, four, or five would do it. They multiply very quickly indeed. In an infected animal the blood would soon be found to swarm with bacilli. He entirely agreed with what Professor M'Fadyean had said.

Evidence for the Defendants.

Mr WINTER, *examined by Mr Willes Chitty*,—said he was the junior partner of the firm of Pattinsons & Winter, millers and corn and cake merchants, carrying on business at Penrith and other places in Cumberland. They had been carrying on business for quite twenty-five years. The firm deals largely in corn, feeding meals, and stuffs for cattle. For upwards of twenty years, to his knowledge, they had dealt largely in this linseed cake purchased from Messrs Earle & King. This was the same sort of cake which was sent to Lord Brougham, of which complaint was now made. In 1899 something like 420 tons of cake of one kind or other was supplied to people in the neighbourhood from Messrs Earle & King. He had no knowledge how much of that was linseed cake. That was linseed and square feed cake. The proportion of linseed cake was probably about one-third. In 1900 they sent out upwards of 520 tons; the proportion of linseed cake probably being about the same. Until this particular event they had never had a complaint with regard to that cake. They still continued to supply it all round the country. The cake is bought from Earle & King. The linseed cake is delivered in bulk; the square feed cake comes in bags. In Lord Brougham's case the cake always went to their warehouse first, because Lord Brougham's carts came for it as it was required, on a written order. There was always a written order for the quantity of

cake, either linseed or square feed cake. They have three different places at Penrith where they keep the cake, the principal warehouse being in connection with their mill, adjoining the mill where the great bulk of it is stored. They have also another warehouse in another part of the town, just for market days. They had been supplying Lord Brougham with food stuffs for nearly twenty years he thought. Before 1899 he thought Lord Brougham might have got small quantities of linseed and feed cake from Earle & King, but about the beginning of 1899 Lord Brougham began to use Earle & King's manufactures more freely. They (Pattinsons & Winter) had nothing to do with the distribution of the cake to the different farms. They had supplied Mr Riley with undecorticated cotton cake manufactured in Glasgow, which had nothing to do with Earle & King and their cake. Since then Mr Riley has frequently had Earle & King's cake, but as a rule he has it supplied by waggon load to Blenkhous Station. At all events this case of anthrax at Mr Riley's which had been referred to had nothing whatever to do with Earle & King's cake. On the 19th of March last Mr Westgarth, Lord Brougham's bailiff, came to their place of business to buy some bran. He asked to see a sample of bran. A sample was sent for and shown to Mr Westgarth. He made no suggestion then against the cakes. The first suggestion that they heard of anything being wrong with the cake was a letter from Messrs Arnison & Co. on the 23rd of April. That was the only complaint they had had from either Lord Brougham, his agent, or anybody in connection with Lord Brougham. They had heard rumours. They knew from the papers, and they also knew because Lord Brougham had ceased to take cakes from them, that something was wrong, but the first intimation was on the 25th of April—a letter delivered by hand from Messrs Arnison's office. He knew Lord Brougham had ceased to take cake at the end of the month. The letter from Messrs Arnison's was at once sent on to Messrs Earle & King, the same day. A day or two afterwards a communication was received from them. At the end of April, as soon as he had their reply, he went to Messrs Arnison's office to get permission to go to the farm. He had a list of a large number of farms all round Penrith to which this stuff was supplied. It was very generally used all round Penrith, and they supplied a large number of farms in Cumberland and Westmoreland. He had a list of places to which the cake was sent from their works, which came from the same bulk as that sent to Lord Brougham. They received no complaint with regard to that cake.

Cross-examined by Mr Shearman,—the witness said that they did not manufacture the cake. For more than twenty years it had been supplied to them by Messrs Earle & King. It is bought from them, and ordered as it is required. Generally speaking, not less than four tons is bought at a time: sometimes they contract for 50 or 100 tons. He had a list of what went into the warehouse where Lord Brougham's cake came from. Lord Brougham's cake was in five or six different orders. All the linseed cake came out of the same warehouse. They had fourteen tons of linseed cake from Earle & King during March, which went into the same warehouse as Lord Brougham's came out of. There were five different consignments from Earle & King; four tons of linseed cake on 16th March, and two tons on 24th March; fourteen cwt. of linseed cake along with eighteen cwt. of seed cake. One consignment cannot be afterwards distinguished from another. He could not tell anything about the ingredients, or how the cake was manufactured.

MR HAROLD KING, examined by Mr Pickford,—said he was one of the Managers of Earle & King, Limited. He attended chiefly to the manufacture. He had been connected with the firm for about twenty-four years. They turn out, roughly speaking, about 500 tons a week of all cakes. About two-thirds of that would be linseed cake. It is distributed all over the

United Kingdom. They have between 500 and 600 customers, some consumers, but chiefly dealers; about 350 dealers and 250 consumers. There had never before been a suggestion of anthrax being found in their cake. For January, February, and March last year they had three consignments of linseed from Calcutta and Kurrachee, all Indian seed. They had none at all from Russia or Turkey, which were the countries mentioned by Professor M'Fadyean. The linseed is partially cleaned when it arrives, but they put it through a process of cleaning before crushing it. This is done with sieves and revolving screens to take all impurities out. It is then rolled between chilled iron rollers which reduces it to a fine meal or flour; it then passes into what they call kettles, made of iron, and 6 feet in diameter and $2\frac{1}{2}$ feet deep; it is there what they call cooked, that is to say, there is a screen constantly revolving through the crushed seed emitting steam at a temperature of 280° F. into the meal. This keeps the meal moving at the same time, and it remains in the kettle about twenty minutes. It is then taken out, put in hydraulic presses, and pressure put on it, and portion of the oil extracted, and what remains is the linseed cake which is used for cattle. When they received the letter from Messrs Pattinsons & Winter, enclosing Messrs Arnison's letter, they wrote to the former and asked to be allowed to make an investigation, preferably on the ground of the outbreak, and to draw samples for analysis. On 4th May, Mr Greville Earle and he went to Penrith. They saw Messrs Arnison, and were not allowed to make an investigation. Finding that they could do nothing there they returned to Liverpool. He then instructed Professor Boyce to come down to their mill, and to take samples of everything and anything he liked, and to subject them to analysis. He took samples of the linseed which the cake was made from, and all other stuff they had in the mill. On 25th May, he again went to Penrith with Professor Boyce and Mr Greville Earle. They went to Eamont Bridge Farm and met Mr Westgarth, and were shown over the premises. Professor Boyce took some samples from the cake that was on the Eamont Bridge Farm. Mr Westgarth told them it was a sample of the same bulk that Professor M'Fadyean had taken. He told them that Professor M'Fadyean's samples had been sent out in a bag that was lying about the place. They were shown the place where the bull that died in 1891 was buried and the one that died in 1900. They went on to the Gardens Farm and took samples from there. They then went on to the Hospital Farm, and found half a ton of cake there, samples of which were taken. After that they received Professor Boyce's report as to the result of his analysis. They instructed him to submit some to Professor Sims Woodhead of Cambridge. They got his report, and were quite satisfied. Subsequently, for the purpose of this case, they got the report from Professor Klein and Professor Muir.

Cross-examined by Mr Shearman,—the witness said that all the linseed they had during January, February, and March, was from India. He was very ignorant about anthrax, and could not say whether India was a country where it was prevalent. They do not always use up all the linseed they have, within a month or two. His reason for saying the linseed ordered before January was used up before this cake was made, was that at that time they were very short of linseed and had a very small stock—the stock was practically nil. Those were the only stocks they got in, and they were all used up. The last of this Indian linseed would be turned into cake towards the end of March. The manufacture goes on every day. The amount of cake turned out during the day depends on how many presses they have working at the linseed. Their utmost capacity for linseed cake would be 70 tons a day and night. Working normally they make only 10 tons a day and night of linseed cake—the amount varies on demand. They get linseed from America and the River Plate, and sometimes from Turkey. They had got it once from

Russia in the last ten years. The kettles vary from 5 to 6 feet in diameter; they are all about the same depth. There is a continuous stream of linseed passing in and out of the kettles. They are steamed at a temperature of 280° F. The temperature of the meal when it leaves the kettle is about 170° F. The steam comes out of a perforated steam stirrer, at the bottom of the kettle. The meal is a fairly solid substance when it is in the kettle; it is not as solid as flour, it is more flocculent. The steam rises through it, and it all gets stirred before it leaves the steam kettle. It comes down past the steam and then passes away. They have no means of testing the average heat of the stuff itself and not of the steam. Considering it leaves the kettle at from 160° to 170° it must be over 100°, or they could not make the cake. He said this was practically their action, as their reputation was at stake.

Re-examined by Mr Pickford,—the witness said that every particle of the meal is exposed to the steam before it leaves the kettle.

PROFESSOR BOYCE, *examined by Mr Wiles Chitty*,—said he was Professor of Pathology and Bacteriology at University College, Liverpool, and Examiner in State Medicine at the University of Oxford, and the Victoria University. He was also Public Analyst for the city of Liverpool. He had had very large experience in conducting investigations with regard to infectious cases, particularly in connection with anthrax, as affecting certain industries. He frequently got samples for analysis for the purpose of ascertaining whether they are infected with bacillus, and particularly with anthrax. He had previously examined many grains, but never seed cake. He had not previously known of anthrax bacillus being found in linseed or linseed cake. In May of last year he was consulted by Messrs Earle & King with reference to this case. He went to their manufactory on the 8th May last year, and made a thorough examination of the various processes concerned in oil cake manufacture, and took numerous samples in sterilised bottles which he bought for the purpose. He took nine samples from the raw grain to the final article, including the oil crushed out at high temperature of the meal in the kettle. He saw the process through which the meal went in the kettle, and the temperature to which it was subjected by means of the steam stirrer. As far as his knowledge went Mr King's account was very correct. He examined the premises and the process of manufacture with the object of ascertaining whether there was any opportunity of the bacillus getting into the cake. There was absolutely nothing to indicate the possibility of it happening at the mill. It would not be expected to occur there, the sources of the grain being known. He afterwards subjected the nine samples to careful analysis. He had samples of the cake in the process of manufacture—of hot cake in various stages. They were all carefully submitted to examination with a view to seeing whether there were any germs of anthrax in them, and there was no trace of anything of the sort. He went to the farms on 25th May in company with Mr King. He there took samples of the cake pointed out by Mr Westgarth, from the Garden Farm, the Hospital Farm, and the Eamont Bridge Farm. One of these was said to be from the same bulk as that taken by Professor M'Fadyean. He subjected that to analysis. He collected his samples himself under special precautions, having known that they were transferred by cart from Messrs Winters to Lord Brougham's estate, and knowing that on the farm they might be subjected to contamination he brought his own material for collecting the samples, and sealing the samples in the presence of the gentlemen who were with him. There was absolutely no opportunity for his to be contaminated after leaving the farm. He experimented on these, and found no anthrax bacilli, though very searching tests were used. As a crucial and final experiment he had a young bull fed with this stuff; having taken the precautions he had mentioned, and having considerable samples of cake, he fed a heifer with the usual

preparation from some he had over, amounting in each instance to 2 lbs. of cake, and the animal had had no ill effect and was still alive. A bull was buried in the yard in 1891. In his opinion it was quite probable that the germs from that bull could infect animals in 1900. He expressed this opinion because the excreta of that animal would have to be taken into consideration. It was not only the carcase but the excreta and the blood which would have contaminated the building, and it would lurk there for years. The germs can be conveyed from one place to another in hundreds of different ways. One of the greatest sources is dust, and they can be conveyed by rats, mice, and other animals. Anthrax is spread in this country by the skinning of animals which have died from that disease. He sent some of the samples to other people, again using precautions; he sent them, simply intimating would they carefully examine the cake and see if the anthrax germ was present. He told them nothing more about the cake. He sent them to Professor Woodhead of Cambridge, Professor Klein in London, and Professor Muir in Glasgow.

Cross-examined by Mr Shearman,—the witness said he took samples of linseed himself from Earle & Kings. These were subjected to the usual bacteriological tests of plain cultivation with gelatine, agar, and bouillon, and by inoculation afterwards. In each experiment he used one gramme of linseed, that is fifteen grains. His experiment would certainly lead to the cultivation of whatever bacteria there was in that meal. Numerous bacteria, such as he would expect, were discovered. He examined them microscopically, and could find no anthrax. He took samples of cake on that occasion also. He obtained colonies of bacteria from the cake, but in very much smaller numbers than from the crude grain. Not a large number of bacteria from the cake survived the boiling process mentioned before. A very large proportion, reckoning it from visible appearance, 99 per cent. of the original bacteria, had been killed in the process of manufacture. In the samples of cake which he took from the warehouse there were very few bacteria. They were bacteria of a definite kind that could be classified. With regard to the samples taken at the farm later on, he attempted to cultivate bacilli from them. In each experiment he used the stock quantity, one gramme. He certainly made a dozen experiments. In the dozen grammes he did not find the anthrax bacillus, but other bacilli. With regard to the samples he took from the farm, he tried the principle of inoculation. He inoculated the most susceptible animal—guinea-pigs. From each sample he inoculated two guinea-pigs. All together he inoculated twelve guinea-pigs, all of which thrived. One guinea-pig would receive the juice of one gramme of the cake, it having been triturated with sterilised broth. There was one experiment for each guinea-pig. One gramme at a time of the cake was pounded up to mix with the broth, and inoculate the guinea-pig. All he could get of that, which was usually 4 cubic centimetres, he used in inoculating one guinea-pig. He had made a study of anthrax. He was not aware of its prevalence in any one locality more than another; he could not answer the question as to whether it was very prevalent in India. It was a great deal more prevalent in some countries than others. He should say that it was not more prevalent in India and the River Plate. He thought it was more prevalent in China. He could not answer as to whether it was prevalent in India; it was in the River Plate. He had had no experience, but he supposed it came over like other grain. There would be no difficulty in the spore or bacillus finding its way through a bag. It was possible that linseed coming over in bags could be infected by spores coming out of hides. Precautions had to be used. He did not agree for an instant with the statement that anthrax is, in most cases, communicated by food or water. If the infection was by the mouth, blood would be found in the ali-

mentary canal. If blood was found in the alimentary canal he would not conclude that the infection came from food. If pathological lesions were seen in the intestines it would be said that it was taken by the food. His theory was that it might be communicated by the food. If he were called in to advise as to the stopping of an outbreak, he would take the ordinary mode of suggesting a change of food. The first matter to which he would direct his inquiry would not be the different foods which the animal had had, nor would he endeavour to eliminate those foods which could be proved to have no infection. He should adopt preliminary precautions which he considered necessary, that was, seeing that the animals were kept in an absolutely pure place where no chance of infection could have arisen. Assuming that he was asked to stop an outbreak, he would not direct his attention only to the food in order to trace the source of the outbreak, but to any source of previous contamination. He would inquire whether there had been any previous contamination. If he found that there had been no disease for the last nine years he should have to examine into that statement. It was possible that foods might have something to do with the matter, but from his experience of grains used for feeding animals he had come to the conclusion that it was improbable. He suggested that in this case the infection arose from the outbreak in 1891, or some possible case of anthrax that might have died, but been unsuspected, and passed away. He thought that the most probable explanation was previous anthrax in the place, and that the bacillus was blowing about. In his opinion, if the cake had been infected after it was delivered to the place, that would bear on this enquiry. He did not doubt that Professor M'Fadyean found anthrax bacillus in the cake. His theory was that that anthrax bacillus must have got on this cake after it got to the farm. Having seen and examined the storeroom personally, and inspected it and its position, he had in his own mind no doubt that if germs were conveyed by wind—and he had no doubt that they were, and that they were conveyed by human beings and smaller animals—the chance of their getting into the place where the cake was stored was great. Some of the bacilli blew in, and if they arrived on the cake, that was to be responsible for the death of the six cattle. The bacillus might also have got there from the hands of the servants who went in. In that case he suggested it would get on the cake in the form of both a spore and a bacillus. He did not agree with Professor M'Fadyean that a spore could not develop except at a temperature of over 60°, going on for many days. He suggested that mere blowing about, or mere touching, would infect a large quantity of cake; he said this from experience with hides; one infected hide may infect a good many bags, and the room in which the bags are stored, as well as linseed if there is any on the same ship with the hides. Assuming that the bacilli got into the room he would reasonably expect that it would contaminate the hay and other kinds of food. If the bacillus was blown in, he should think it strange if it did not affect the water and the other articles of food on the premises. He could offer no suggestion as to how the infection which killed the cow at Gardens Farm got there.

Re examined by Mr Pickford,—the witness said that if it was known how these things happened, it would be easier to prevent them. The whole point of infectious diseases was that it could not always be told how the infection carried. Supposing it to be suggested that the infection had got into the linseed, and the bacillus was on the linseed, and then that survived the treatment with fans and steam, he would certainly expect to find the bacillus in the body of the cake, having seen the process of manufacture. In his experiments, he experimented with the body of the cake, excluding the outside. If it had been carried as he suggested was possible on the farm, he would expect to find it on the outside. Professor M'Fadyean had not excluded the outside of the cake from his evidence. If the bacillus had

been carried as he suggested possible, that would account for the difference between his experiments and Professor M'Fadyean's. If it were in the linseed, which was made into the body of the cake, he should find it very difficult to account for the difference. With regard to the question of whether an outbreak could be traced to something which happened nine or ten years ago, he could give no dates on record of cases in which they had been treated in that way. He believed that Professor Koch could do that, but he could not do so himself from his own experience. It was most certainly possible for the men who had skinned and opened the first bull that died, even after washing their hands and disinfecting their boots, to be a source of contagion. His experience in hospital practice had shown him that washing the hands in 99 cases out of 100 was useless, unless it was seen to be properly done, taking antiseptic precautions. Infection can readily be carried by the class of men on a farm. The fact that the two men who skinned and opened the carcass continued to attend on and feed the cattle afterwards might certainly account for the subsequent cases. The bacteria which he found in the cake were harmless.

Professor SIMS WOODHEAD, *examined by Mr Pickford*,—said that he was Professor of Pathology at the University of Cambridge. He received four samples of cake from Professor Boyce. Each piece of cake was about from 8 to 9 inches square—perhaps 6 inches square. They were sent to him with a request to see whether there was any anthrax bacillus. He was not told whether he was to find it or not. He made the examination in the ordinary recognised way. He repeated his experiment on four different occasions, and never found a trace of the anthrax bacillus. If the linseed were infected and then passed through the process described, he would expect to find it throughout the cake, outside and inside. If it had been communicated in any way accidentally, as had been suggested, it would be found outside only. He thought it possible there might be spores or bacilli resulting from a case that happened nine years before. He had spores which he had kept for eleven years which were still vital.

Cross-examined by Mr Shearman,—the witness said that it was possible that the 1891 outbreak had something to do with it, but it was difficult to say what were the probabilities. He should say that spores could live 100 years. He should not like to say the exact sizes of the four pieces of cake supplied to him. He used up the whole of them in the different experiments. He thought he had inoculated sixteen guinea pigs, eight rats, and eight mice, and he had fed sixteen guinea pigs. He found no anthrax bacillus in any of them; some of them he killed and examined the blood. He had no doubt that the cakes supplied to him had no anthrax bacillus in them. If he were asked to inquire into an outbreak of anthrax, he should certainly direct his attention particularly to the food the animals had had. It was his belief that anthrax was frequently communicated along with the food, much more often than otherwise with regard to cattle. In the early stage of proceedings, with regard to cattle, he would first look at the food. It is undoubtedly possible to eliminate all other foods which are above suspicion if they are supplied to other animals with no bad results. He had not the slightest doubt that Professor M'Fadyean found the bacillus; the question was whether it got on to the cake after it got to the farm. That was the point at issue. The fact that a cow died at a farm half a mile off, did not, he thought, prove that it broke out in the food. In all cases of outbreak of anthrax it was most difficult to trace the original cause; one frequently found there had been unsuspected causes. There was always some doubt in every disease in knowing exactly to what the outbreak was due. He should not like to offer any specific explanation of the outbreak at the Gardens Farm, unless he knew all the conditions of the case. He thought that it was quite probable

that other articles of food besides the cake might have been infected in the same way. Had they been examined and nothing found, he should have thought it extraordinary. If the cake was infected after it came to the farm, he would have expected that there was an equal chance of everything else being infected. In these experiments he found other bacilli, although harmless. He was rather struck by the fact of the small number of organisms, and was anxious to know how in seed cake it was so small, but when it was explained that it was subjected to high temperature it was obvious that most of the organisms had been killed off.

Re-examined by Mr Pickford,—the witness said there was nothing remarkable in finding bacilli in these substances. There were very few without them, especially those in a liquid form, and where a body temperature was kept up for any length of time it would be extraordinary if they were not there. Supposing the bacillus came with the linseed, he could not understand the immunity that was possessed by all the other farms which had been supplied from the same bulk. That was the difficulty, in his mind, and a very great difficulty, in explaining it as the result of the linseed cake that there should be five or six on one farm, and at the other farm, where the same cake was used, there should be no cases at all; that seemed to him a very extraordinary thing. It would be explained if the contamination got to the cake after it came to the farm. He could see no other explanation if it was proved to be from the linseed cake.

Dr E. E. KLEIN, *examined by Mr Willes Chitty*,—said he was a Fellow of the Royal Society. He had had a good deal of experience in the examination of bacteria poisoning. He received four samples of oil cake for analysis from Professor Boyce. He was instructed to analyse them for anthrax, nothing more. He made the examination, which was, comparatively speaking, elaborate. He found no traces of anthrax in any of the four samples. He thought if there had been any trace of anthrax he would have discovered it.

Cross-examined by Mr Shearman,—the witness said he examined all four samples. Each sample was sent in a sterile tube, and the amount was roughly between five and ten grammes. The whole of each sample was used for analysis. He could not use more, because he did not get more.

Professor MUIR, *examined by Mr Pickford*,—said he was Professor of Pathology in the University of Glasgow. He received four test tubes full of small fragments of cake. He had rather more than Dr Klein had. He used it all in his examination, and found no anthrax bacillus.

Cross-examined by Mr Shearman,—the witness said he got these samples from Professor Boyce.

Professor JOHN PENBERTHY, *examined by Mr Pickford*,—said he was a Fellow of the Royal College of Veterinary Surgeons, London. He was Professor of Medicine at the Royal Veterinary College. He had not made any examination of these samples. He had had a very considerable experience in anthrax. From his experience, considering what happened in 1891, if there was a case of anthrax and the animal skinned and opened before being buried, and was moved from one place to another, that would, in 1900, remain a source of great danger. The spores might remain from the excreta in the shippen or have been left in the paddock. Lots of things might occur to retain the existence of the poison there; even smaller animals may contract the disease and die, and keep up generations in that way. The spores are known to live longer under favourable conditions than nine or ten years. It was quite impossible for him, or anyone else, to say how frequently these favourable conditions did exist, but they knew from practical experience that sometimes they did exist, even in a state of nature. Assuming that the linseed was contaminated as suggested, he could not in any way explain the immunity of all the other places supplied with the cake. He could not conceive a dis-

tribution of the anthrax germ among a large quantity of linseed cake which would have limited its action, and so distributed it on Lord Brougham's estate. He could not theorise mentally as to how such a thing could possibly occur, knowing what he did of the stirring up of the linseed and the different consignments of cake, and knowing how cake is usually treated on a farm, and how it is taken from the store and delivered out to the farm where there are nearly forty animals. He had heard what had happened with the first animal which died in 1891: that was skinned and opened by men who were told to wash their hands and disinfect their boots, and afterwards attended to the feeding and care of their cattle. He thought that was a possible and most probable source of infection; but beyond that it was well known that during the life of the animal, that was while it was suffering from anthrax, it was then discharging anthrax germs in the different discharges of the body, and so the place where the animal stood would become contaminated, and any person who went into that place at the time the animal was suffering from the disease might have contaminated his boots or any other part unwittingly, nobody suspecting that he was doing it, and would carry it not only to a farm half a mile off, but from Rome to London. If a man who was attending to a sick cow or bull was also feeding the other cows he might leave some of the germs, which would not be eaten till three weeks afterwards, and he might have walked to the Gardens Farm on the night of the 9th, which would have come into effect on the 25th. He could not, of course, possibly account for the immunity that there was at the Hospital Farm. There was not much difference in the susceptibility of young animals above a certain age and old ones.

Cross-examined by Mr Shearman,—the witness said that, with regard to the 1891 outbreak, he had known spores to live for nine or ten years. He had kept the bacilli in tubes. In the museum special precautions were not used. They are put under conditions suitable for their surviving. He did not know whether a nine-year-old spore would be considered very old; he did not pose as an expert on bacteriology. His business had been to investigate practically on farms and elsewhere, to trace the origin of anthrax germs, and to try to eliminate that source of danger. He agreed with Professor M'Fadyean that it was possible an outbreak might happen after nine years. He believed that in every country during the last ten or fifteen years the Authorities had paid great attention to anthrax. The Government Authorities consult eminent gentlemen, and get the best opinion the gentlemen can give probably. The study of anthrax was one of the oldest of the bacteriological studies. There was also a practical study of it, and statistics were taken of the outbreaks and their cause. He knew that during the past year the average number of animals in an outbreak was less than two. That had not precluded more than two animals dying in more than one place. There were 577 deaths last year, and in a large number of cases there was only one death proved. Sometimes there was a large outbreak, and a large number of animals died, but the average number of animals that died was only two. With regard to the outbreak of 1891, he saw the place where the animal was buried. He heard that the place was fenced round, and that cattle had no access for grazing. That was a reasonable and proper place to bury it in, but he should have preferred a place further off from where animals were kept. If it was buried 6 inches without the quicklime and putrefaction took place, that which was buried was not likely to become a spore. If the spores did turn up in 1891, if they were there before the carcase was buried, there was a possibility that they came out of the carcase buried 7 feet deep in quicklime. The spore must have come from something contaminated by the carcase. He had suggested it as probable that it was from something contaminated in 1891, and he had suggested that the spore was lurking about and was dormant for

nine years, and he had then suggested other means which would bring on fresh generations. He had heard that there were several animals which had died. He did not suggest rats. Other animals had died and their deaths had been entered in the diary. The causes of death were not extremely explicit, and there was a possibility even that mistakes might have occurred, because it was his professional opinion that it was common for death from anthrax to be overlooked and not suspected, and that was the opinion of other experts. He said that there was a very strong possibility of cases of anthrax having occurred on this establishment between 1891 and 1900 which nobody but himself had suspected, inasmuch as the cause of the deaths of animals had not been explained to him. There was one animal in 1898, the death of which had not been properly explained; there were others which had not been properly explained to him, for instance, muco-enteritis.

Re-examined by Mr Pickford,—the witness said that he thought muco-enteritis was a general term which did not carry the cause of the disease which brought about the death. When an animal was buried the bacillus was very soon disposed of by putrefaction. He attached much importance to the fact that this animal was skinned and opened before burial, and millions of spores were probably formed. Professor M'Fadyean, in giving evidence in another case, had said that where an animal dies of anthrax, and its blood is shed on the ground, a hot temperature develops the spores which may have lain in the soil for a 100 years. He did not put it as high as that. He had great respect for Professor M'Fadyean's opinion. It was in 1847 that it was first suggested that any such thing existed. They simply lie waiting their opportunity, and nothing happens to them until they are taken in by an animal grazing over the spot. He knew so much about farm operations that if anyone told him that the men did not go near the burial place he would not believe it. He considered that implements were very facile things for communicating the germs.

Mr JAMES HUDSON, *examined by Mr Willes Chitty*,—said that he was agent for Lord Lonsdale, and resided near Penrith. There are several farms there belonging to the Earl of Lonsdale. He had known the Defendants in business for over twenty years. During that time they had purchased feeding stuff from them. They had been in the habit of purchasing this "E. & K." linseed cake. From December 1899 to the end of March 1900 they had purchased a good deal more than 20 tons. They had not had a case of anthrax, and he had nothing to say with regard to the cake. Probably 400 cattle were fed on the linseed cake at a time.

Cross-examined by Mr Shearman,—the witness said the cake was supplied in different quantities as required. In January they had it fifteen times, seven times in February, and ten times in March. None of the cattle suffered any ill effects; during the whole time they had not had a single case of illness. Since he had been Lord Lonsdale's agent there had never been a case of anthrax on the farm.

Mr JOHN CASTLEHOW TOPPIN, *examined by Mr Pickford*,—said he lived at Musgrave Hall, Penrith. His farm is about 400 acres in his own hands. For twenty years, more or less every year, he had purchased this "E. & K." linseed cake from the Defendants, Messrs Pattinsons & Winter. On 14th February 1900, he purchased 14 cwt.; and on the 19th, 4 cwt. On the 19th March, 15 cwt.; and on the 23rd, 14 cwt. He had never had a case of anthrax in his life. As a rule, he feeds from 100 to 400 head of cattle on this stuff, but this year the linseed cake was used for feeding up young bulls for sale, so he had only about fifty.

Mr WILLIAM GRISENTHWAITE, *examined by Mr Willes Chitty*,—said he was County Councillor for Cumberland and a railway contractor. He had a

farm at Penrith. In February and March last year he had thirty cattle. He had been in the habit of purchasing feed stuff for the cattle from Messrs Pattinsons & Winter. On the 26th March last year he purchased some of this linseed cake. He had had a supply of this for a long time before, and fed his cattle on it. He had never had cause to complain of the cake, nor had he had a case of anthrax. He still used the cake.

Cross-examined by Mr Shearman,—the witness said the last supply of cake before 26th March was not from Pattinsons & Winter. He had 5 cwt of "E. & K." cake on 26th March. The last supply before that was from Messrs Grains & Co., and was not "E. & K." cake.

Re-examined by Mr Pickford,—the witness said that that was the first lot of "E. & K." cake he had. He had none before March of last year. He had these two lots, and they were all right.

Mr JUSTICE MATHEW, in summing up, said that in this case the contract in respect of which the action was brought was admitted. The Defendants agreed that they were bound to supply this linseed cake fit for the consumption of animals, but they said that if the animals suffered in this case it was not because their contract was broken, not because the linseed when delivered was infectious in character; and that if any damage was done to the animals by the linseed cake it was due to something that had occurred after the cake had been delivered, and therefore was a matter for which they were not responsible. It was for the Plaintiff to satisfy the Jury, as reasonable men, that the loss of the cattle was due to the cause to which he had attributed it. They were not to act on suspicion, nor upon any theory that did not appear to them to be entirely satisfactory, but were to use the intellects with which they were fortunately provided for their guidance in the daily affairs of life, and say whether the fair inference from the evidence laid before them was or was not that the Defendants were liable. Certainly it could not be denied that the Defendants started with a strong presumption in favour of the cake, because it appeared that the manufacturers had been producing this cake for years, selling it extensively, and selling it in this district, and no single complaint had ever been made of the presence of this bacillus, or of any consequent loss of cattle by anthrax. The evidence of experts about that was that it was a curious circumstance with reference to this disease that it was casual and capricious in its character, and that was shown by what had occurred on this farm itself. The food which had been said to be contaminated was supplied to all these animals, and only a few out of the whole herd had been affected in this way. So that it was quite possible it might not affect particular cattle, and there might not be a case of this sort: but it by no means showed that in one consignment there might not be these infected elements. That theory alone, would not, of course, displace the body of evidence they had heard on the other side. But, unquestionably, it was a strong point in the Defendants favour, and had been dealt with with great ability by Mr Pickford, who represented them. But, of course, he had to go further. He had to step away, it was said, from the hard facts which were before them, to theories, and this theory, it was said, was so far fetched that they ought not to go upon it for a moment, namely that before that, nine years before, an animal suffering from anthrax died on this farm. It had been treated at once, although apparently opposed as they heard, and apparently skinned before the disease was ascertained, it had been treated in the old hary way and subjected to the suspicion of the Local Authorities, and every precaution taken—precautions which were really found sufficient to prevent the spread of the disease. The animal was buried, the burial place was fenced off and used as a receptacle for garden refuse, and for many more and for many long years there had been no more of the disease on this farm. Then, unfortunately, the two

animals were suddenly seized and died within a few hours. It had been said that the explanation lay in the fact that nine years before one animal had died of the same disease. The bacillus under favourable conditions of temperature would develop into a number of spores. It was not yet clear how long the spores would live; they might be dormant for nine years or longer, and then be blown about and inhaled by an animal, and so the disease might be contracted. But it was said for the Plaintiffs, supposing the evidence offered by the scientific witnesses on the other side to be correct, it was only theory without a shadow of proof, and ought not to be adopted by the jury. It had been asked what instance could be given of a case where under similar circumstances, where an animal was buried as in this case, the disease broke out after two, three, four, or five years, much less nine? Not a single instance of the kind was known. Then it was said it was a mere theory and that it was impossible for them to adopt it, and if they did not adopt it something else must have been the cause. The theory was that these two animals had been infected by spores and the way they had been dealt with explained all that occurred subsequently. One of these animals had been partially dressed and skinned; and there was at once, it was said, the probability that some blood had escaped, and blood was, no doubt, of all other things, the source of infection in a disease of this sort, and, although precautions had been taken in the presence of the Authorities, which were considered sufficient at the time, it was possible—according to some witnesses it was probable—that the workmen might have carried away on their hands some particle of blood from the process they had gone through with these animals, and that this particle might have found its way to this cake that they were handling and to other articles of food, and so it was suggested that they were contaminated.

It was further said that the same workman carried a quantity of cake to the other farm, to the Gardens Farm, and in each case left behind from his hand traces of blood which would explain all that subsequently happened. The Defendants thus suggested that the disease was contracted from something that had been put on the cake, and something which was not found in it. Experts had been called in, who had examined the cake on the part of the Defendants, and, although not the slightest imputation was cast on the sufficiency and propriety of their experts, no bacillus was found in anything that they examined. These gentlemen who were called in as scientific witnesses had this great comfort, and great security, that as a general rule they were only expressing opinions, not like ordinary witnesses who came to tell about facts. That allowed them considerable elbow-room when they got into the witness-box. Mr Boyce was the strongest witness for the Defendant on this part of the case, and he thought at first it was "very probable" that the disease could have been communicated from something that touched the surface of the cake; but when a little bit pressed "probable" became "possible," and he was the one witness who really put that forward as a likely explanation of what occurred. They had heard a very able discussion from the Counsel for the Defendants of the evidence offered on their behalf, and if they thought that created any reasonable doubt as to the cause of the disease, they would no doubt be entitled to their verdict. On the other hand, if they came to the conclusion that it was made out satisfactorily to them as reasonable men that there was no other cause than the condition of this cake when it was delivered, the Plaintiff would be entitled to their verdict.

After a short absence from the Court, the Jury found a verdict for the Plaintiff, and judgment was given for £304 13s. 6d., being the amount of damages agreed upon.

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THE COMBATING OF TUBERCULOSIS IN THE LIGHT
OF THE EXPERIENCE THAT HAS BEEN GAINED
IN THE SUCCESSFUL COMBATING OF OTHER
INFECTIOUS DISEASES.¹

By Geh. Med.-Rath Professor Dr ROBERT KOCH, Direktor des
Instituts für Infektions Krankheiten in Berlin.

THE task with which this Congress will have to busy itself is one of the most difficult, but it is also one in which labour is most sure of its reward.

I need not point again to the innumerable victims tuberculosis annually claims in all countries, nor to the boundless misery it brings on the families it attacks. You all know that there is no disease which inflicts such deep wounds on mankind as this. All the greater, however, would be the general joy and satisfaction if the efforts that are being made to rid mankind of this enemy, which consumes its inmost marrow, were crowned with success.

There are many, indeed, who doubt the possibility of successfully combating this disease, which has existed for thousands of years, and has spread all over the world. This is by no means my opinion. This is a conflict into which we may enter with a surely founded prospect of success, and I will tell you the reasons on which I base this conviction.

Only a few decades ago the real nature of tuberculosis was unknown to us; it was regarded as a consequence, as the expression, so to

¹ An address delivered at the British Congress on Tuberculosis, London, July 1901.

speak, of social misery, and as this supposed cause could not be got rid of by simple means, people relied on the probable gradual improvement of social conditions, and did nothing. All this is altered now. We know that social misery does indeed go far to foster tuberculosis, but the real cause of the disease is a parasite—that is, a visible and palpable enemy, which we can pursue and annihilate, just as we can pursue and annihilate other parasitic enemies of mankind.

Strictly speaking, the fact that tuberculosis is a preventible disease ought to have become clear as soon as the tubercle bacillus was discovered, and the properties of this parasite and the manner of its transmission became known. I may add that I, for my part, was aware of the full significance of this discovery from the first, and so will everybody have been who had convinced himself of the causal relation between tuberculosis and the tubercle bacillus. But the strength of a small number of medical men was inadequate to the conflict with a disease so deeply rooted in our habits and customs. Such a conflict requires the co-operation of many, if possible of all, medical men, shoulder to shoulder with the State and the whole population; but now the moment when such co-operation is possible seems to have come. I suppose there is hardly any medical man now who denies the parasitic nature of tuberculosis, and among the non-medical public too the knowledge of the nature of the disease has been widely propagated.

Another favourable circumstance is that success has recently been achieved in the combating of several parasitic diseases, and that we have learned from these examples how the conflict with pestilences is to be carried on.

The most important lesson we have learned from the said experience is that it is a great blunder to treat pestilences uniformly. This was done in former times; no matter whether the pestilence in question was cholera, plague, or leprosy: isolation, quarantine, useless disinfection were always resorted to. But now we know that every disease must be treated according to its own special individuality, and that the measures to be taken against it must be most accurately adapted to its special nature, to its etiology. We are entitled to hope for success in combating tuberculosis only if we keep this lesson constantly in view. As so extremely much depends just on this point, I shall take the liberty to illustrate it by several examples.

The pestilence which is at this moment in the foreground of interest, the bubonic plague, may be instructive to us in several respects.

People used to act upon the conviction that a plague patient was in the highest degree a centre of infection, and that the disease was transmitted only by plague patients and their belongings. Even the most recent international agreements are based on this conviction. Although, as compared with formerly, we now have the great advantage that we can, with the aid of the microscope and of experiments on animals, recognise every case of plague with absolute certainty, and although the prescribed inspection of ships, quarantine, the isolation of patients, the disinfection of infected dwellings and ships, are carried out with the utmost care, the plague has, nevertheless, been transmitted everywhere, and has in not a few places

assumed grave dimensions. Why this has happened we know very well, owing to the experience quite recently gained as to the manner in which the plague is transmitted. It has been discovered that only those plague patients that suffer from plague-pneumonia—a condition which is fortunately infrequent—are centres of infection, and that the real transmitters of the plague are the rats. There is no longer any doubt that, in by far the majority of the cases in which the plague has been transmitted by ocean traffic, the transmission took place by means of plague among the ship rats. It has also been found that wherever the rats were intentionally or unintentionally exterminated, the plague rapidly disappeared; whereas at other places, where too little attention had been paid to the rat plague, the pestilence continued. This connection between the human plague and the rat plague was totally unknown before, so that no blame attaches to those who devised the measures now in force against the plague if the said measures have proved unavailing. It is high time, however, that this enlarged knowledge of the etiology of the plague be utilised in international as well as in other traffic. As the human plague is so dependent on the rat plague, it is intelligible that protective inoculation and the application of antitoxic serum have had so little effect. A certain number of human beings may have been saved from the disease by that, but the general spread of the pestilence has not been hindered in the least.

With cholera the case is essentially different; it may, under certain circumstances, be transmitted directly from human beings to other human beings, but its main and most dangerous propagator is water, and, therefore, in the combating of cholera, water is the first thing to be considered. In Germany, where this principle has been acted on, we have succeeded for four years in regularly exterminating the pestilence (which was introduced again and again from the infected neighbouring countries) without any obstruction of traffic.

Hydrophobia too is not void of instruction for us. Against this disease the so-called protective inoculation proper has proved eminently effective as a means of preventing the outbreak of the disease in persons already infected, but, of course, such a measure can do nothing to prevent infection itself. The only real way of combating this pestilence is by compulsory muzzling. In this matter also we have had the most satisfactory experience in Germany, but have at the same time seen that the total extermination of the pestilence can be achieved only by international measures, because hydrophobia, which can be very easily and rapidly suppressed, is always introduced again year after year from the neighbouring countries.

Permit me to mention only one other disease, because it is etiologically very closely akin to tuberculosis, and we can learn not a little for the furtherance of our aims from its successful combating. I mean leprosy. It is caused by a parasite which greatly resembles the tubercle bacillus. Just like tuberculosis, it does not break out till long after infection, and its course is almost slower. It is transmitted only from person to person, but only when they come into close contact, as in small dwellings and bedrooms. In this disease, accordingly, immediate transmission plays the main part; transmission by animals, water, or the like is out of the question. The

combative measures, accordingly, must be directed against this close intercourse between the sick and the healthy. The only way to prevent this intercourse is to isolate the patients. This was most rigorously done in the Middle Ages by means of numerous leper-houses, and the consequence was that leprosy, which had spread to an alarming extent, was completely stamped out in Central Europe. The same method has been adopted quite recently in Norway, where the segregation of lepers has been ordered by a special law. But it is extremely interesting to see how this law is carried out. It has been found that it is not at all necessary to execute it strictly, for the segregation of only the worst cases, and even of only a part of these, sufficed to produce a diminution of leprosy. Only so many infectious cases had to be sent to the leper-houses that the number of fresh cases kept regularly diminishing from year to year. Consequently the stamping-out of the disease has lasted much longer than it would have lasted if every leper had been inexorably consigned to a leper-house, as in the Middle Ages; but in this way too the same purpose is gained, slowly indeed, but without any harshness.

These examples may suffice to show what I am driving at, which is to point out that in combating pestilences we must strike at the root of the evil, and must not squander force in subordinate ineffective measures. Now the question is whether what has hitherto been done, and what is about to be done against tuberculosis really strikes at the root of tuberculosis, so that it must sooner or later die.

In order to answer this question it is necessary first and foremost to inquire how infection takes place in tuberculosis. Of course, I presuppose that we understand by tuberculosis only those morbid conditions which are caused by the tubercle bacillus.

In by far the majority of cases of tuberculosis the disease has its seat in the lungs, and has also begun there. From this fact it is justly concluded that the germs of the disease, *i.e.*, the tubercle bacilli, must have got into the lungs by inhalation. As to the question where the inhaled tubercle bacilli have come from, there is also no doubt. On the contrary, we know with certainty that they get into the air with the sputum of consumptive patients. This sputum, especially in advanced stages of the disease, almost always contains tubercle bacilli, sometimes in incredible quantities. By coughing, and even speaking, it is flung into the air in little drops, *i.e.*, in a moist condition, and can at once infect persons who happen to be near the coughers. But then it may also be pulverised when dried, in the linen or on the floor for instance, and get into the air in the form of dust.

In this manner a complete circle, a so-called *circulus vitiosus*, has been formed for the process of infection, from the diseased lung, which produces phlegm and pus containing tubercle bacilli, to the formation of moist and dry particles (which, in virtue of their smallness, can keep floating a good while in the air), and finally to new infection, if particles penetrate with the air into a healthy lung and originate the disease anew. But the tubercle bacilli may get to other organs of the body in the same way, and thus originate other forms of tuberculosis. This, however, is a considerably rarer case. The sputum of consumptive people, then, is to be regarded as the main source of the infection of tuberculosis. On this point, I suppose, all

are agreed. The question now arises whether there are not other sources too, copious enough to demand consideration in the combating of tuberculosis.

Great importance used to be attached to the hereditary transmission of tuberculosis. Now, however, it has been demonstrated by thorough investigation that, though hereditary tuberculosis is not absolutely non-existent, it is nevertheless extremely rare, and we are at liberty, in considering our practical measures, to leave this form of origination entirely out of account.

But another possibility of tubercular infection exists, as is generally assumed, in the transmission of the germs of the disease from tubercular animals to man. This manner of infection is generally regarded nowadays as proved, and as so frequent that it is even looked upon by not a few as the most important, and the most rigorous measures are demanded against it. In this Congress also the discussion of the danger with which the tuberculosis of animals threatens man will play an important part. Now, as my investigations have led me to form an opinion deviating from that which is generally accepted, I beg your permission, in consideration of the great importance of this question, to discuss it a little more thoroughly.

Genuine tuberculosis has hitherto been observed in almost all domestic animals, and most frequently in poultry and cattle. The tuberculosis of poultry, however, differs so much from human tuberculosis that we may leave it out of account as a possible source of infection for man. So, strictly speaking, the only kind of animal tuberculosis remaining to be considered is the tuberculosis of cattle, which, if really transferable to man, would indeed have frequent opportunities of infecting human beings through the drinking of the milk and the eating of the flesh of diseased animals.

Even in my first circumstantial publication on the etiology of tuberculosis I expressed myself regarding the identity of human tuberculosis and bovine tuberculosis with reserve. Proved facts which would have enabled me sharply to distinguish these two forms of the disease were not then at my disposal, but sure proofs of their absolute identity were equally undiscoverable, and I therefore had to leave this question undecided. In order to decide it, I have repeatedly resumed the investigations relating to it, but so long as I experimented on small animals, such as rabbits and guinea pigs, I failed to arrive at any satisfactory result, though indications which rendered the difference of the two forms of tuberculosis probable were not wanting. Not till the complaisance of the Ministry of Agriculture enabled me to experiment on cattle, the only animals really suitable for these investigations, did I arrive at absolutely conclusive results. Of the experiments which I have carried out during the last two years along with Professor Schütz, of the Veterinary College in Berlin, I will tell you briefly some of the most important.

A number of young cattle which had stood the tuberculin test, and might therefore be regarded as free from tuberculosis, were infected in various ways with pure cultures of tubercle-bacilli taken from cases of human tuberculosis; some of them got the tubercular sputum of consumptive patients direct. In some cases the tubercle-bacilli or the sputum were injected under the skin, in others into the peritoneal cavity, in others into the jugular vein. Six animals were

fed with tubercular sputum almost daily for seven or eight months; four repeatedly inhaled great quantities of bacilli, which were distributed in water, and scattered with it in the form of spray. None of these cattle (there were nineteen of them) showed any symptoms of disease, and they gained considerably in weight. From six to eight months after the beginning of the experiments they were killed. In their internal organs not a trace of tuberculosis was found. Only at the places where the injections had been made small suppurative foci had formed, in which few tubercle bacilli could be found. This is exactly what one finds when one injects dead tubercle bacilli under the skin of animals liable to contagion. So the animals we experimented on were affected by the living bacilli of human tuberculosis exactly as they would have been by dead ones; they were absolutely insusceptible to them.

The result was utterly different, however, when the same experiment was made on cattle free from tuberculosis with tubercle bacilli that came from the lungs of an animal suffering from bovine tuberculosis. After an incubation-period of about a week the severest tubercular disorders of the internal organs broke out in all the infected animals. It was all one whether the infecting matter had been injected only under the skin or into the peritoneal cavity or the vascular system. High fever set in, and the animals became weak and lean; some of them died after a month and a half to two months, others were killed in a miserably sick condition after three months. After death extensive tubercular infiltrations were found at the place where the injections had been made, and in the neighbouring lymphatic glands, and also far advanced alterations of the internal organs, especially the lungs and the spleen. In the cases in which the injection had been made into the peritoneal cavity the tubercular growths which are so characteristic of bovine tuberculosis were found on the omentum and peritoneum. In short, the cattle proved just as susceptible to infection by the bacillus of bovine tuberculosis as they had proved insusceptible to infection by the bacillus of human tuberculosis. I wish only to add that preparations of the organs of the cattle which were artificially infected with bovine tuberculosis in these experiments are exhibited in the Museum of Pathology and Bacteriology.

An almost equally striking distinction between human and bovine tuberculosis was brought to light by a feeding experiment with swine. Six young swine were fed daily for three months with the tubercular sputum of consumptive patients. Six other swine received bacilli of bovine tuberculosis with their food daily for the same period. The animals that were fed with sputum remained healthy and grew lustily, whereas those that were fed with the bacilli of bovine tuberculosis soon became sickly, were stunted in their growth, and half of them died. After three months and a half the surviving swine were all killed and examined. Among the animals that had been fed with sputum no trace of tuberculosis was found, except here and there little nodules in the lymphatic glands of the neck, and in one case a few grey nodules in the lungs. The animals, on the other hand, which had eaten bacilli of bovine tuberculosis had, without exception (just as in the cattle experiment), severe tubercular diseases, especially tubercular infiltration of the greatly enlarged lymphatic glands of the

neck and of the mesenteric glands, and also extensive tuberculosis of the lungs and the spleen.

The difference between human and bovine tuberculosis appeared not less strikingly in a similar experiment with asses, sheep, and goats, into whose vascular systems the two kinds of tubercle bacilli were injected.

Our experiments, I must add, are not the only ones that have led to this result. If one studies the older literature of the subject, and collates the reports of the numerous experiments that were made in former times by Chauveau, Günther and Harms, Bollinger, and others, who fed calves, swine, and goats with tubercular material, one finds that the animals that were fed with the milk and pieces of the lungs of tubercular cattle always fell ill of tuberculosis, whereas those that received human material with their food did not. Comparative investigations regarding human and bovine tuberculosis have been made very recently in North America by Smith, Dinwiddie, and Frothingham, and their result agreed with that of ours. The unambiguous and absolutely conclusive result of our experiments is due to the fact that we chose methods of infection which exclude all sources of error, and carefully avoided everything connected with the stalling, feeding, and tending of the animals that might have a disturbing effect on the experiments.

Considering all these facts, I feel justified in maintaining that human tuberculosis differs from bovine, and cannot be transmitted to cattle. It seems to me very desirable, however, that these experiments should be repeated elsewhere, in order that all doubt as to the correctness of my assertion may be removed.

I wish only to add that, owing to the great importance of this matter, the German Government has appointed a commission to make further inquiries on the subject.

But, now, how is it with the susceptibility of man to bovine tuberculosis? This question is far more important to us than that of the susceptibility of cattle to human tuberculosis, highly important as that is too. It is impossible to give this question a direct answer, because, of course, the experimental investigation of it with human beings is out of the question. Indirectly, however, we can try to approach it. It is well known that the milk and butter consumed in great cities very often contain large quantities of the bacilli of bovine tuberculosis in a living condition, as the numerous infection-experiments with such dairy products on animals have proved. Most of the inhabitants of such cities daily consume such living and perfectly virulent bacilli of bovine tuberculosis, and unintentionally carry out the experiment which we are not at liberty to make. If the bacilli of bovine tuberculosis were able to infect human beings, many cases of tuberculosis caused by the consumption of alimenta containing tubercle bacilli could not but occur among the inhabitants of great cities, especially the children. And most medical men believe that this is actually the case.

In reality, however, it is not so. That a case of tuberculosis has been caused by alimenta can be assumed with certainty only when the intestine suffers first—*i.e.*, when a so-called primary tuberculosis of the intestine is found. But such cases are extremely rare. Among many cases of tuberculosis examined after death, I myself

remember having seen primary tuberculosis of the intestine only twice. Among the great *post-mortem* material of the Charité Hospital in Berlin ten cases of primary tuberculosis of the intestine occurred in five years. Among 933 cases of tuberculosis in children at the Emperor and Empress Frederick's Hospital for Children, Baginsky never found tuberculosis of the intestine without simultaneous disease of the lungs and the bronchial glands. Among 3104 *post-mortems* of tubercular children, Biedert observed only sixteen cases of primary tuberculosis of the intestine. I could cite from the literature of the subject many more statistics of the same kind, all indubitably showing that primary tuberculosis of the intestine, especially among children, is a comparatively rare disease, and of these few cases that have been enumerated, it is by no means certain that they were due to infection by bovine tuberculosis. It is just as likely that they were caused by the widely propagated bacilli of human tuberculosis, which may have got into the digestive canal in some way or other—for instance, by swallowing saliva of the mouth. Hitherto nobody could decide with certainty in such a case whether the tuberculosis of the intestine was of human or of animal origin. Now we can diagnose them. All that is necessary is to cultivate in pure culture the tubercle-bacilli found in the tubercular material, and to ascertain whether they belong to bovine tuberculosis by inoculating cattle with them. For this purpose I recommend subcutaneous injection, which yields quite specially characteristic and convincing results. For half a year past I have occupied myself with such investigations, but, owing to the rareness of the disease in question, the number of the cases I have been able to investigate is but small. What has hitherto resulted from this investigation does not speak for the assumption that bovine tuberculosis occurs in man.

Though the important question whether man is susceptible to bovine tuberculosis at all is not yet absolutely decided, and will not admit of absolute decision to-day or to-morrow, one is nevertheless already at liberty to say that, if such a susceptibility really exists, the infection of human beings is but a very rare occurrence. I should estimate the extent of infection by the milk and flesh of tubercular cattle, and the butter made of their milk, as hardly greater than that of hereditary transmission, and I therefore do not deem it advisable to take any measures against it.

So the only main source of the infection of tuberculosis is the sputum of consumptive patients, and the measures for the combating of tuberculosis must aim at the prevention of the dangers arising from its diffusion. Well, what is to be done in this direction? Several ways are open. One's first thought might be to consign all persons suffering from tuberculosis of the lungs whose sputum contains tubercle bacilli to suitable establishments. This, however, is not only absolutely impracticable, but also unnecessary. For a consumptive who coughs out tubercle bacilli is not necessarily a source of infection on that account, so long as he takes care that his sputum is properly removed and rendered innocuous. This is certainly true of very many patients, especially in the first stages, and also of those who belong to the well-to-do classes, and are able to procure the necessary nursing. But how is it with people of very small means? Every medical man who has often entered the dwellings of the poor, and I

can speak on this point from my own experience, knows how sad is the lot of consumptives and their families there. The whole family have to live in one or two small, ill-ventilated rooms. The patient is left without the nursing he needs, because the able-bodied members of the family must go to their work. How can the necessary cleanliness be secured under such circumstances? How is such a helpless patient to remove his sputum, so that it may do no harm? But let us go a step further and picture the condition of a poor consumptive patient's dwelling at night. The whole family sleep crowded together in one small room. However cautious he may be, the sufferer scatters the morbid matter secreted by his diseased lungs every time he coughs, and his relatives close beside him must inhale this poison. Thus whole families are infected. They die out, and awaken in the minds of those who do not know the infectiousness of tuberculosis the opinion that it is hereditary, whereas its transmission in the cases in question was due solely to the simplest processes of infection, which do not strike people so much, because the consequences do not appear at once, but generally only after the lapse of years.

Often, under such circumstances, the infection is not restricted to a single family, but spreads in densely inhabited tenement houses to the neighbours, and then, as the admirable investigations of Biggs have shown in the case of the densely peopled parts of New York, regular nests or foci of disease are formed. But, if one investigates these matters more thoroughly, one finds that it is not poverty *per se* that favours tuberculosis, but the bad domestic conditions under which the poor everywhere, but especially in great cities, have to live. For, as the German statistics show, tuberculosis is less frequent, even among the poor, when the population is not densely packed together, and may attain very great dimensions among a well-to-do population when the domestic conditions, especially as regards the bedrooms, are bad, as is the case, for instance, among the inhabitants of the North Sea coast. So it is the overcrowded dwellings of the poor that we have to regard as the real breeding-places of tuberculosis; it is out of them that the disease always crops up anew, and it is to the abolition of these conditions that we must first and foremost direct our attention if we wish to attack the evil at its root, and to wage war against it with effective weapons.

This being so, it is very gratifying to see how efforts are being made in almost all countries to improve the domestic conditions of the poor. I am also convinced that these efforts, which must be promoted in every way, will lead to a considerable diminution of tuberculosis. But a long time must elapse ere essential changes can be effected in this direction, and much may be done meanwhile in order to reach the goal much more rapidly.

If we are not able at present to get rid of the danger which small and overcrowded dwellings involve, all we can do is to remove the patients from them, and, in their own interests and that of the people about them, to lodge them better; and this can be done only in suitable hospitals. But the thought of attaining this end by compulsion of any kind is very far from me; what I want is that the consumptives may be enabled to obtain the nursing they need better than they can obtain it now. At present a consumptive in an advanced stage of the disease is regarded as incurable and as an unsuitable inmate for a

hospital. The consequence is that he is reluctantly admitted and dismissed as soon as possible. The patient, too, when the treatment seems to him to produce no improvement, and the expenses, owing to the long duration of his illness, weigh heavily upon him, is himself animated by the wish to leave the hospital soon. That would be altogether altered if we had special hospitals for consumptives, and if the patients were taken care of there for nothing, or at least at a very moderate rate. To such hospitals they would willingly go; they could be better treated and cared for there than is now the case. I know very well that the execution of the project will have great difficulties to contend with, owing to the considerable outlay it entails. But very much would be gained if, at least in the existing hospitals, which have to admit a great number of consumptives at any rate, special wards were established for them, in which pecuniary facilities would be offered them. If only a considerable fraction of the whole number of consumptives were suitably lodged in this way, a diminution of infection and consequently of the sum-total of tuberculosis could not fail to be the result. Permit me to remind you in this connection of what I said about leprosy. In the combating of that disease also great progress has already been made by lodging only a fair number of the patients in hospitals. The only country that possesses a considerable number of special hospitals for tubercular patients is England, and there can be no doubt that the diminution of tuberculosis in England, which is much greater than in any other country, is greatly due to this circumstance. I should point to the founding of special hospitals for consumptives and the better utilisation of the already existing hospitals for the lodging of consumptives as the most important measure in the combating of tuberculosis, and its execution opens a wide field of activity to the State, to municipalities, and to private benevolence. There are many people who possess great wealth, and would willingly give of their superfluity for the benefit of their poor and heavily afflicted fellow-creatures, but do not know how to do this in a judicious manner. Here is an opportunity for them to render a real and lasting service by founding Consumption Hospitals or purchasing the right to have a certain number of consumptive patients maintained in special wards of other hospitals free of expense.

As, however, unfortunately, the aid of the State, the municipalities, and rich benefactors will probably not be forthcoming for a long time yet, we must for the present resort to other measures that may pave the way for the main measure just referred to, and serve as a supplement and temporary substitute for it.

Among such measures I regard obligatory notification as specially valuable. In the combating of all infectious diseases it has proved indispensable as a means of obtaining certain knowledge as to their state, especially their dissemination, their increase, and decrease. In the conflict with tuberculosis also we cannot dispense with obligatory notification; we need it not only in order to inform ourselves as to the dissemination of this disease, but mainly in order to learn where help and instruction can be given, and especially where the disinfection which is so urgently necessary when consumptives die or change their residence has to be effected. Fortunately it is not at all necessary to notify all cases of tuberculosis, nor even all cases of consumption, but only those that, owing to the domestic conditions, are sources of

danger to the people about them. Such limited notification has already been introduced in various places, in Norway, for instance, by a special law, in Saxony, by a ministerial decree, in New York and in several American towns, which have followed its example. In New York, where notification was optional at first and was afterwards made obligatory, it has proved eminently useful. It has thus been proved that the evils which it used to be feared the introduction of notification for tuberculosis would bring about need not occur, and it is devoutly to be wished that the examples I have named may very soon excite emulation everywhere.

There is another measure, closely connected with notification, viz., disinfection, which, as already mentioned, must be effected when consumptives die or change their residence, in order that those who next occupy the infected dwelling may be protected against infection. Moreover, not only the dwellings but also the infected beds and clothes of consumptives ought to be disinfected.

A further measure, already recognised on all hands as effective, is the instructing of all classes of the people as to the infectiousness of tuberculosis, and as to the best way of protecting oneself. The fact that tuberculosis has considerably diminished in almost all civilised States of late is attributable solely to the circumstance that knowledge of the contagious character of tuberculosis has been more and more widely disseminated, and that caution in intercourse with consumptives has increased more and more in consequence. If better knowledge of the nature of tuberculosis has alone sufficed to prevent a large number of cases, this must serve us as a significant admonition to make the greatest possible use of this means, and to do more and more to bring it about that everybody may know the dangers that threaten him in intercourse with consumptives. It is only to be desired that the instructions may be made shorter and more precise than they generally are, and that special emphasis be laid on the avoidance of the worst danger of infection, which is the use of bedrooms and small ill-ventilated workrooms simultaneously with consumptives. Of course the instructions must include directions as to what consumptives have to do when they cough and how they are to treat their sputum.

Another measure, which has come into the foreground of late, and which at this moment plays to a certain extent a paramount part in all efforts for the combating of tuberculosis, works in quite another direction. I mean the founding of sanatoria for consumptives.

That tuberculosis is curable in its early stages must be regarded as an undisputed fact. The idea of curing as many tubercular patients as possible in order to reduce the number of those that reach the infectious stage of consumption, and thus to reduce the number of fresh cases, was therefore a very natural one. The only question is whether the number of persons cured in this way will be great enough to exercise an appreciable influence on the retrogression of tuberculosis. I will try to answer this question in the light of the figures at my disposal.

According to the business report of the German Central Committee for the Establishment of Sanatoria for the Cure of Consumptives, about 5500 beds will be at the disposal of these institutions by the end of 1901, and then, if we assume that the average stay of each patient will be three months, it will be possible to treat at least 20,000 patients

every year. From the reports hitherto issued as to the results that have been achieved in the establishments we learn further that about 20 per cent. of the patients that have tubercle bacilli in their sputum lose them by the treatment there. This is the only sure test of success, especially as regards prophylaxis. If we make this the basis of our estimates, we find that 4000 consumptives will leave these establishments annually as cured. But, according to the statistics ascertained by the German Imperial Office of Health, there are 226,000 persons in Germany over fifteen years of age who are so far gone in consumption that hospital-treatment is necessary for them. Compared with this great number of consumptives the success of the establishments in question seems so small that a material influence on the retrogression of tuberculosis in general is not yet to be expected of them. But pray do not imagine that I wish, by this calculation of mine, to oppose the movement for the establishment of such sanatoria in any way. I only wish to warn against the over-estimating of their importance which has recently been observable in various quarters, based apparently on the opinion that the war against tuberculosis can be waged by means of sanatoria alone, and that other measures are of subordinate value. In reality the contrary is the case. What is to be achieved by the general prophylaxis resulting from recognition of the danger of infection and the consequent greater caution in intercourse with consumptives is shown by a calculation of Cornet's regarding the decrease of mortality from tuberculosis in Prussia in the years 1889 to 1897. Before 1889 the average was 31.4 per 10,000, whereas in the period named it sank to 21.8, which means that, in that short space of time, the number of deaths from tuberculosis was 184,000 less than was to be expected from the average of the preceding years. In New York, under the influence of the general sanitary measures directed in a simply exemplary manner by Biggs, the mortality from tuberculosis has diminished by more than 35 per cent. since 1886. And it must be remembered that both in Prussia and New York the progress indicated by these figures is due to the first beginnings of these measures. Considerably greater success is to be expected of their further development. Biggs hopes to have got so far in five years that in the city of New York alone the annual number of deaths from tuberculosis will be 3000 less than formerly. I take this opportunity of most urgently recommending Dr Biggs' organisation to the study and imitation of all municipal sanitary authorities.

Now, I do indeed believe that it will be possible to render the sanatoria considerably more efficient. If strict care be taken that only patients be admitted for whom the treatment of those establishments is well adapted, and if the duration of the treatment be prolonged, it will certainly be possible to cure 50 per cent., and perhaps still more. But even then, and even if the number of the sanatoria be greatly increased, the total effect will always remain but moderate. The sanatoria will never render the other measures I have mentioned superfluous. If their number become great, however, and if they perform their functions properly, they may materially aid the strictly sanitary measures in the conflict with tuberculosis.

If now, in conclusion, we glance back once more to what has been done hitherto for the combating of tuberculosis and forward to what has still to be done, we are at liberty to declare with a certain satisfac-

tion that very promising beginnings have already been made. Among these I reckon the consumption hospitals of England, the legal regulations regarding notification in Norway and Saxony, the organisation created by Biggs in New York, the sanatoria, and the instruction of the people. All that is necessary is to go on developing these beginnings, to test, and if possible to increase their influence on the diminution of tuberculosis, and wherever nothing has yet been done, to do likewise.

If we are continually guided in this enterprise by the spirit of genuine preventive medical science, if we utilise the experience gained in conflict with other pestilences, and aim, with clear recognition of the purpose and resolute avoidance of wrong roads, at striking the evil at its root, then the battle against tuberculosis, which has been so energetically begun, cannot fail to have a victorious issue.

TUBERCLE BACILLI IN COWS' MILK AS A POSSIBLE SOURCE OF TUBERCULOUS DISEASE IN MAN.¹

By JOHN M'FADYEAN, M.B., M.R.C.V.S., Royal Veterinary College, London.

AS recently as a few days ago, when I was mentally arranging the material for the paper which I have now the great honour of submitting to this Congress, I was under the impression that it would not be necessary to formally prove that the term tuberculosis as it is now employed by medical men and veterinary surgeons relates to one and the same disease. I thought that I might ask my audience to accept it as proved, and generally admitted, that tuberculosis in man is caused by a single definite species of organism—the tubercle bacillus; that this organism is also the cause of the disease to which veterinary surgeons apply the term tuberculosis in the case of cattle and other domesticated species, and that there therefore existed a *prima facie* case against the germs formed in the bodies of tuberculous animals as a possible source of tuberculous disease in human beings.

To-day, however, the position of anyone who undertakes to discuss the intercommunicability of human and bovine tuberculosis is very different from what it would have been a week ago, for in the interval the greatest living authority on tuberculosis—the world renowned discoverer of the tubercle bacillus, and the man to whom we are mainly indebted for our knowledge of the cause of tuberculosis—has declared his conviction that human and bovine tuberculosis are practically two distinct diseases. I do not know how far the reasons assigned by Dr Koch for the opinion which he now holds on this question may have commended themselves to the members of this Congress, and I am overwhelmed at finding myself in a position which compels me to offer some criticism on the pronouncement of one the latchet of whose shoes I am not worthy to unlace.

That bovine and human tuberculosis are identical diseases was generally supposed to have been finally determined by Dr Koch himself, when he discovered that the human and the bovine germs

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contained bacilli that were identical in morphological, tinctorial, and cultural characters, and showed that the artificial cultures from both sources produced indistinguishable effects when they were employed to infect a variety of animals. The labours of hundreds of workers during the succeeding eighteen years produced nothing in serious conflict with the conclusion that human and bovine tuberculosis were identical diseases, but they brought to light what appeared to be additional evidence of this identity, such as the discovery that tuberculin produced a specific reaction in tuberculous cattle, whether human or bovine bacilli had been employed in its preparation. In short, the identity of the bacilli from the two sources appeared to be as firmly established as any other generally accepted opinion regarding the identity or non-identity of bacteria associated with disease in more than one species of animal. Since it thus appeared to be proved that the only difference between human and bovine tubercle bacilli lay in their accidentally different position—one being parasitic in man and the other in cattle—it was natural to conclude that, when circumstances were favourable for the transference of bacilli from one species to the other, human tuberculosis might have an animal origin, and *vice versa*.

Opinions varied as to the frequency with which this transmission of tuberculosis from one species to the other occurred, but practically never within the last eighteen years regarding the possibility and probability of such reciprocal infection. What are the grounds upon which we are asked to discard convictions that appeared to rest on such a solid basis? I shall endeavour to state them briefly, as I understand Dr Koch's train of reasoning.

(1) The bacilli found in cases of bovine tuberculosis are much more virulent for cattle and other domestic quadrupeds than the bacilli found in cases of human tuberculosis.

(2) This difference is so marked and so constant that it may be relied upon as a means of distinguishing the bacilli of bovine tuberculosis from those of the human disease, even assuming that the former may occasionally be found as a cause of disease in man.

(3) If bovine bacilli are capable of causing disease in man, there are abundant opportunities for the transference of the bacilli from the one species to the other, and cases of primary intestinal tuberculosis from the consumption of tuberculous milk ought to be of common occurrence. But *post-mortem* examination of human beings proves that cases of primary intestinal tuberculosis are extremely rare in man, and therefore it must be concluded that the human subject is immune against infection with the bovine bacilli, or is so slightly susceptible that it is not necessary to take any steps to counteract the risk of infection in this way.

Now, with the utmost diffidence I venture to submit that at least one of the premises contained in this argument is not well founded, that the others have little or no bearing on the question, and that there still remain reasonable grounds for regarding tuberculous cows' milk as distinctly dangerous to human beings.

It cannot be denied that what may be called bovine tubercle bacilli are as a rule distinctly more virulent for cattle and other domesticated animals than human bacilli, or that the results of experiments indicate that in natural circumstances there is little danger of cattle becoming

infected from human beings. But it cannot be admitted that the low virulence of human bacilli for cattle proves, or even makes it probable, that bovine bacilli have only a feeble pathogenic power for man. That might have been held to be probable if it had been shown that bovine bacilli were very virulent only for cattle, but since it is well established that these bacilli are highly dangerous for such diverse species as the rabbit, horse, dog, pig, and sheep, and, in short, for almost every quadruped on which they have been tried, it appears to be highly probable that they are also dangerous to man. At any rate it is impossible to cite any ascertained fact relating to other bacterial diseases that makes the contrary conclusion probable. It is well known that the majority of disease-exciting bacteria are harmful to only one or two species, but all those that are common to all the domesticated animals are also pathogenic to man.

With regard to the view that the difference between human and bovine bacilli in respect of virulence for cattle is of such a fixed and constant character that it may be relied upon to distinguish the one from the other, it need only be said that that is very far from proved. It appears to be quite possible that what may be called the normal or average virulence of bovine bacilli for cattle may be reduced by passage through the human subject. Besides, there are very great differences in the virulence of tubercle bacilli found in animals of the same species, and if a low degree of virulence for cattle is to be taken as the distinguishing feature of human bacilli there will be no difficulty in proving that the human disease is sometimes transmitted to the lower animals.

The third proposition in Dr Koch's argument is the only one which is really germane to the point at issue, viz., that only cases of primary intestinal tuberculosis can possibly have had their origin in infected milk or meat, and that "such cases are extremely rare." Dr Koch refers to several large series of *post-mortem* observations that appear to justify this statement, and adds that he could have cited many more pointing to the same conclusion. Now, if it were a fact that all the statistics relating to this point were unanimous, it would have to be admitted that primary intestinal tuberculosis is rare in the human subject, and that cases of infection through milk are still rarer, though even then it might be advisable to take measures to prevent the few cases. But the statistics are not by any means unanimous, and those that are likely to appeal with most force to the people of this country are not at all in accord with those quoted from Germany. During the last few years the evidence obtainable from the *post-mortem* records of two of the largest hospitals for children in this country have been analysed with great care, in order to see what evidence they afforded as to the relative frequency of the different methods of infection in tuberculosis. In the case of the Hospital for Sick Children in Great Ormond Street this has been done by Dr George Still, and in the case of the Royal Hospital for Sick Children in Edinburgh by Dr Shennan. The conclusion at which Dr Still arrived was that in 29.1 per cent. of the cases of tuberculosis in children primary infection appeared to have taken place through the intestine. That is very far from being an insignificant proportion, and it is a striking fact that Dr Shennan arrived at an almost identical conclusion, and estimated that 28.1 per cent. of the cases of

tuberculosis among children in Edinburgh are due to alimentary infection. There does not appear to be any ground for supposing that there is a large margin of error in these statistics, as the number of cases dealt with was considerable (547 in the two series), and in both series the *post-mortem* appearances were interpreted in a way to which no exception can be taken. In face of these statistics it is not possible to assent to the statement that cases of primary tuberculosis of the alimentary canal are extremely rare. Precisely the contrary conclusion is the one that must in the meanwhile be drawn with regard to the state of affairs in this country, viz., that, at least in children, primary infection by way of the alimentary canal is comparatively common.

I therefore submit that there is still a strong *prima facie* case against animal tuberculosis as a possible source of human tuberculosis, and it becomes necessary to consider whether there are any data from which one may estimate the extent of the danger to which human beings are exposed through the occurrence of tubercle bacilli in milk.

The evidence in favour of the view that the ingestion of tuberculous milk is one of the causes of human tuberculosis includes a number of recorded cases in which the relationship of cause and effect appeared to be obvious. From the nature of the circumstances, evidence of this kind is very scanty, and it must be admitted that very few of the alleged examples are absolutely convincing. Tuberculosis is a disease that develops slowly, and assuming for the moment that tubercle bacilli do occur in milk, and are a cause of disease in persons consuming such milk, it is obvious that, as a rule, the very act by which the infection is brought about destroys the only direct evidence of cause and effect that exists.

One could only expect to be able to trace the disease to the milk when, after the onset of symptoms pointing to infection by way of the mouth, the cow from which the milk had been obtained was still available for examination. In practice this is rarely the case, and it is therefore not surprising that medical literature contains very few specific instances of the infection of human beings with tuberculosis by means of milk. It is obvious, however, that the entire absence of evidence of this kind would in no way exonerate milk from the suspicion of being one of the causes of human tuberculosis.

We have already seen that, at least in this country, in a considerable number of cases of tuberculosis occurring in early life, the first seeds of the disease appear to have entered the body by way of the mouth. What proportion of these cases ought to be ascribed to tubercle-infected milk? It scarcely appears to be possible to give a very confident reply to this question, though some distinguished authorities have not hesitated to express the opinion that practically all the cases of primary intestinal tuberculosis occurring in childhood may be set down to this cause. The late Sir Richard Thorne Thorne, in the Harben Lectures on the administrative control of tuberculosis, which he delivered in 1898, expressed his conviction that tuberculous milk was the main cause of *tabes mesenterica* in children, and he characterised the loss of child life from this cause as appalling. The evidence on which this formidable charge was laid against the milch cow was of the following nature. The Registrar-General's Returns

show that during the last fifty years there has been a marked decline in the death-rate from human phthisis, which is the form that tuberculosis generally takes when the bacilli are inhaled. On the other hand, during the same period there has been only a slight decline in the death-rate at all ages from that form of tuberculosis which is ascribable to alimentary infection, and among children under one year of age there have been a notable increase in the mortality from that form of the disease. The decline in the death-rate from phthisis is ascribable to the great improvements which have been effected during the last fifty years in the hygiene of human habitations, such as improvements in lighting, drainage, and ventilation. These, naturally, have not interfered with infection through milk, which has therefore remained unchecked, and in infants has even increased, because during the last fifty years cows' milk has entered more largely into the dietary of very young children.

There are several weak points in this argument. Perhaps the weakest of all is the assumption that the deaths certified under the head of *tabes mesenterica* correspond closely with those which the pathologist would classify as cases of primary alimentary infection. It is scarcely possible to doubt that the term *tabes mesenterica* in the Registrar-General's Returns covers a heterogeneous collection of cases, of which the majority may not be cases of tuberculosis at all. But even if it is agreed to accept all the cases registered under the head of *tabes mesenterica* as instances of primary alimentary infection, the figures found in the Registrar-General's Returns do not support the contention that milk is responsible for all the cases of *tabes*. It is true that they indicate an increase in the death-rate from alimentary tuberculosis among children under twelve months old, but, on the other hand, there appears to have been a considerable decline in the death-rate from the same cause at all ages between one and five years. Now, if tuberculous milk were a frequent cause of tuberculosis, one would not have expected the death-rate from that cause to decline among children between one and five years of age, for there is no reason to suppose that there has been any decline in the use of cows' milk in the feeding of children at that age during the last fifty years. The fact appears to be that the Registrar-General's Returns do not afford much trustworthy information with regard to the number of cases of primary alimentary tuberculosis, and are absolutely worthless as an indication of the extent to which human beings are infected by means of milk.

There is another direction in which one may turn for evidence on this point. We cannot with any pretence to accuracy ascertain the number of persons that annually become infected by milk, but we may be able to form some estimate of the existing danger in this connection by collecting information as to the frequency with which milk contains tubercle bacilli. We know that about 30 per cent. of all the cows giving milk in this country are tuberculous in some degree. This statement no doubt indicates a deplorable state of affairs, but in the present connection it is not quite so alarming as it at first sight appears. Fortunately not every cow that is tuberculous gives milk containing tubercle bacilli. It is true that opinions with regard to this point are not absolutely unanimous, but there is ample evidence to justify the assertion that as a rule the milk is not danger-

ous until the udder itself becomes diseased. The experiments pointing to an opposite conclusion form only a small minority, and the results obtained in most of them were probably due to carelessness on the part of the experimenter. In a few of the cases in which the milk of an apparently healthy udder was found to be infective, it is probable that the gland tissue was in reality diseased, though not to an extent discoverable without microscopic examination. The important question, therefore, is not what proportion of milch cows are tuberculous, but what proportion of them have tuberculous udders. Some authorities have estimated this to be as high as 10 per cent., but the proportion is certainly much less than that in Great Britain. My own experience leads me to think that about 2 per cent. of the cows in the milking herds in this country are thus affected. Now, the milk secreted by a tuberculous udder always contains tubercle bacilli, and it sometimes contains enormous numbers of them, and when these facts are apprehended one begins to realise the seriousness of the danger to which, in the present state of affairs, those who drink uncooked milk are exposed. But there are one or two considerations that make the danger greater than the mere statement of the number of cows affected would at first sight indicate. In the first place, the udder disease is not attended by any pain or tenderness in milking, and the milk for a considerable time after the udder has become manifestly diseased may appear quite wholesome, though in reality it is charged with the germs of tuberculosis. It therefore often happens that the gravity of the condition is not realised by the milker or the owner of the cow, and the milk continues to be sold for human consumption. There is scarcely any room for doubt that if it were sold and consumed unmixed with other milk, some of the persons partaking of it would become infected. In practice it is usually mixed with the milk from other cows that have healthy udders, and thus the germs are distributed among a larger number of persons. Even tuberculous milk that has been thus much diluted may prove infective, but the danger to the individual consumer is in inverse proportion to the degree of dilution.

Since about one cow in fifty is the subject of tuberculosis of the udder, and the average number of cows in the milking herds of this country is less than fifty, it follows that the majority of dairies and farms supply milk that is free from tubercle bacilli, or at least does not contain any derived from this source. On the other hand, when the infective material is present it operates with the greatest intensity in the milk of single cows, and in the mixed milk from small herds.

It must be added that tuberculous disease of the udder is not the only source of tubercle bacilli found in milk. A great deal of the milk in the market contains a considerable quantity of dust and dirt, most of which comes from the cow's udder and the hands of the milker, and part from the dust of the air of the cowshed. When 30 per cent. of the cows in a byre are tuberculous, the dirt in that building, and the atmosphere in it, are almost certain to contain tubercle bacilli, and some of these are very likely to find their way into the milk. The more dirt milk contains, the greater is the chance that tubercle bacilli from that source may be present.

What has been said with regard to the extent of the danger to which the public are exposed through the sale of milk containing

tubercle bacilli may be summed up as follows: The danger cannot be defined by stating how many persons are thus infected annually, or what fraction the persons thus infected form of the total number who contract tuberculosis in the course of a year. At the same time, it is impossible to doubt that the danger is a very real one, since at the present time milk is a vehicle by which tubercle bacilli are often introduced into the bodies of human beings.

Means of Averting the Danger.

The ideal method of counteracting this source of human disease would be to stamp out bovine tuberculosis, or to prevent the sale of milk from every cow that is tuberculous. Unfortunately, it must be admitted that at present that is unattainable. At the present time probably not less than 30 per cent. of all the breeding and milking cattle in this country are in some degree affected with tuberculosis, and to urge that the disease should be attacked on the lines adopted in dealing with cattle plague and pleuro-pneumonia is an effectual method of preventing any Government from touching the subject. But, although the complete and rapid extermination of the disease is impossible, it does not follow that nothing can be done, or ought to be done, in the way of prevention. The disease has attained to its present alarming proportions simply because, until quite recently, altogether erroneous notions were held regarding its cause, and because there has hitherto been the most absolute neglect of the precautions necessary for its prevention. The greatest obstacle to successful action against tuberculosis, whether in man or animals, is the ignorance of the laity regarding the cause of the disease. The immense majority of cattle-owners are not yet convinced that contagion is the only cause of tuberculosis, and very few of them have yet made the slightest effort to check the spread of the disease. As a rule, cows and other cattle visibly ill from tuberculosis are still left alive and in close association with their fellows, although the lowest grade of common-sense and prudence would suggest that such animals ought to be promptly killed, or, at least, isolated. It is not want of common-sense, nor is it mainly lack of means, that is responsible for this inaction; it is simply a want of conviction on the part of cattle-owners that tuberculous animals are dangerous to their companions.

The first thing necessary in this connection is education of the people regarding the nature of the disease. It is necessary, because in this country, where Parliament never moves except by the force of public opinion, the legislative action required will not be taken unless the people are satisfied of its wisdom, and also because even the most drastic sanitary measures enforced by the law are likely to fail if they are not supplemented by the intelligent co-operation of the people. The National Association for the Prevention of Consumption and the Royal Agricultural Society have been endeavoring to disseminate sound views regarding the cause of bovine tuberculosis among farmers and others, but much remains to be done in this direction. But it is not reasonable to ask that things should be done as they are until the education of the farmer in the matter of tuberculosis has been finished. If there are any practical and reasonable measures by which, figuratively speaking, the flow of infection can

from tuberculous cows to healthy human beings can be stopped or impeded, they ought to be immediately enforced.

As soon as the valuable diagnostic properties of tuberculin had been proved by experience, it occurred to a good many people that its assistance ought to be called in in order to exclude tuberculous cows from milking herds. In other words, it was thought that although it might not be practicable to insist upon the application of the tuberculin test to all infected herds, and to compel the isolation or slaughter of all cattle thus found to be infected, it might still be possible to require that only cows found to be free from the disease by the application of the test should be kept for milch purposes. I doubt whether anyone who is well acquainted with the circumstances of the case now believes this practicable. Here, again, the fact that one-third of the cows now giving milk are tuberculous is an insurmountable obstacle. The cost of carrying out the tuberculin test several times annually in all the milking herds in this country would be enormous, and the exclusion of all reacting cows from such herds would seriously disorganise cattle-breeding as well as milk production. Moreover, to rely blindly on the tuberculin test, and to pronounce the milk of every cow that does not react to it free from tubercle bacilli, would be very unsafe. The test is recognised to be one of great value, but it is not infallible. Rather serious defects in connection with it are: (1) that for a period after infection—a period that is sometimes very considerable—an animal will not react; (2) that in some advanced cases of tuberculosis no distinct reaction is obtainable; and (3) that in a considerable proportion of cases a second reaction is not obtainable for some days or weeks after the first. It is therefore clear that if we wish to exclude the milk of tuberculous cows, or if the object is the more restricted one of preventing the sale of milk from tuberculous udders, some system of inspection is necessary. This was the conclusion at which the Second Royal Commission on Tuberculosis arrived. We have already seen that whatever danger attaches to milk comes mainly from the cows with tuberculosis of the udder, and the public health would be almost entirely safeguarded from this danger if we could exclude such animals from our dairies. Periodic examination by competent inspectors would go a long way to securing this object, but the inspection would require to be at rather short intervals, for a tuberculosis of the udder may come into existence and attain most dangerous dimensions in a period of a few weeks. The more frequent the inspection the better, but, of course, this means a great deal of expense.

If every town and rural district produced its own milk, it would be a comparatively simple problem to organise and carry out a fairly efficient system of inspection of milch cows; but as the law at present stands, the majority of the population cannot obtain this safeguard. With the exception of Glasgow, Manchester, and a few other places, a local authority has no power to inspect cows outside its own district, and the helpless position in which this state of the law leaves the inhabitants of London and other large towns is obvious. If cows of which the milk is sold for human food had everywhere to be submitted to periodic inspection, such inspection would naturally be undertaken by the various local authorities, each of which would supervise the cows and cowsheds in its own district; but the com-

pulsory inspection of all the milch cows in the country would be a large undertaking, and perhaps it would be premature to press for it. In the meantime, a good case can be made out for making general the special powers relating to inspection of cows in outside districts which a few fortunate cities have acquired by special Acts of Parliament. This was also one of the directions in which the members of the Second Royal Commission on Tuberculosis considered immediate action to be necessary.

There remain for consideration some other safeguards which would doubtless be less effective than those just discussed, but which, unlike these, would not be difficult to enforce, viz. : (1) compulsory notification of udder disease and of any symptoms of tuberculosis in milch cows, with, of course, the power to inflict a considerable fine for not reporting ; and (2) the interdiction of the sale of milk from any cow suffering from tuberculous disease of the udder, or exhibiting clinical signs of tuberculosis. Against the demand for the amendment of the existing law to the extent of granting the public these very reasonable safeguards against infection through milk it cannot be urged that they would be very expensive, or that they would press harshly on private interests. The present state of the law, or rather the almost entire absence of any law, dealing with tuberculous udder disease in cows is a scandal and a reproach to civilisation. It scarcely sounds credible, but it is a fact that the owner of a cow in the most advanced state of tuberculosis, and exhibiting the most manifest signs of udder disease, may sell that cow's milk for human food as long as the sale has not been specially interdicted on the certificate of a veterinary surgeon, and no penalty attaches to this crime of deliberately or carelessly placing on the market a food material charged with the germs of a dangerous disease.

In the interests of public health, the sale of milk from tuberculous udders, and from cows that are obviously tuberculous in any part of the body, must be stopped, and it must be declared illegal to keep such animals alive. There need be no hesitation in pressing for this reform, because the measures demanded are in the interests of the owners of cattle, and would be advisable even if it were established that bovine tuberculosis is not transmissible to man. There is no dispute as to the danger of visibly tuberculous animals to others of their own species, and it is the very reverse of a hardship to the owner of such animals to insist on their being slaughtered.

It would probably be regarded as a serious omission if I did not refer to one other method of counteracting whatever danger at present attaches to impure milk as a cause of human tuberculosis. No matter how highly charged milk may be with tubercle bacilli, it can be deprived of all danger from that source by raising it to the temperature known to be fatal to these germs. Less than the boiling temperature (212° F.) suffices for this purpose ; but, unfortunately, the lowest temperature that can be relied upon imparts to the milk a flavour that many people find distasteful. That objection does not hold good in the case of infants and young children, and the custom of boiling or steaming the nursery milk for a few minutes cannot be made too general. But while abstinence from uncooked milk is a sure way of avoiding infection with bacilli present in that article of food, it cannot for a moment be admitted that this absolves public health authorities

from all concern with the subject. Arsenical beer may be made harmless by adding the proper antidote before drinking it, but the most courageous brewer would not plead this as an excuse for selling the impure article.

In conclusion, I would venture to express the earnest hope that the Congress will not endorse the view that it is inadvisable to take any measures to prevent the transmission of tuberculosis from the lower animals to human beings. To justify the introduction of measures to that end it is not necessary to contend that this is a common method of infection, or that the danger arising from milk can for a moment be compared with that present in human sputum. The inhalation of tubercle bacilli expelled from the bodies of human patients is doubtless the great cause of human tuberculosis, and every practicable means of preventing infection in that way ought to be employed; but, at the same time, we ought not to concede to the milkman the right to sell us tubercle bacilli, even if we were assured that—like Dr Koch's experimental pigs—we had nothing to fear beyond the development of "little nodules here and there in the lymphatic glands" of our necks and "a few grey tubercles" in our lungs.

TUBERCULOSIS A PARASITIC DISEASE AND ANALOGIES.

By THOS. B. GOODALL, F.R.C.V.S., Christchurch, Hants.

IN the late part of last year I was asked by my esteemed friend, Prof. Hobday, to prepare a paper to be read at the Congress of the National Society for the Prevention of Tuberculosis. I begged of him to be excused (1) because I thought it would be better if the papers were prepared by experts, and the discussion carried on by those interested in their work; and (2) because my ideas on the subject were not in exact conformity with the *popular scientific* opinions of the day.

But I was over-persuaded, and promised a paper, the rough notes of which I had scribbled out, when another letter from the secretary informed me that it had been decided to alter the mode of procedure at the Congress by dropping out some of the papers, and asking my consent to allow mine to stand down, which I gave most readily, for the reasons stated above, particularly the second one.

As events have turned out, I am almost sorry that the paper was not read, because I venture to think that it contains some points that might assist in directing our thoughts to the right solution of the problem set to the scientific world by Professor Koch, and I have felt it to be a duty to place my poor rough notes on record, which I am enabled to do through the courtesy and kindness of the editor of this Journal.

I make no pretence of giving expression to new facts, or of being able to throw new light on the subject of tuberculosis. I merely submit for consideration a few thoughts of a very ordinary everyday hard-working country practitioner—thoughts culled and gathered by observance on the everyday round, but which seem to me to have a practical bearing on the subject which is now engaging the attention of the whole scientific world.

It is not many men who have the opportunity, or, given this, the ability to carry out expensive experiments in the laboratory, and so we outsiders look up to the privileged few, and accept their deductions as the basis on which we meet and fight disease. But there is a laboratory (Nature's great workshop) which is unconditionally free for all, and in which absolutely trustworthy work is being carried on unceasingly, from which any man may form sound deductions, if he will only make a proper use of the faculties which are his God-given heritage. "There is a book who runs may read" is a saying as applicable to science as theology, and the deductions so gained, are, if I may say so, as valuable in arriving at a right conclusion as are the necessarily narrower experiments of the pure scientist.

But put together the laboratory work of the scientist and facts deduced from an observation of Nature's unchanging laws, and we may arrive at a possible solving of the truth we are all intent on seeking.

We may take it now as a fact beyond dispute that all communicable diseases are *parasitic*—that is, they are all due to living organisms living on or in the body of their host, and by their presence interfering with the normal functions of one or of several of the organs of that host—and the terms "contagion" and "infection" signify simply that these parasitic organisms may be conveyed from the body of one "host" to that of another; but from Nature's great laboratory we learn that, as regards the more highly organised parasites—pediculi, acari, etc.—each order, family, and even variety of the larger animals has its parasite of a particular variety, which is peculiar to itself, and they are very rarely indeed interchanged.

If we carry our reasoning still further to the relationship of parasites to the great classes of the vertebrates, we shall be astonished to find how very rarely their diseases, which are practically all parasitic, are intercommunicable.

It will, of course, be understood that I am not including in this category those diseases induced by internal intestinal and other parasites, which pass a part of the cycle of their existence in one animal, and the other in an animal that, as a rule, preys on its former host, as, for example, tape-worm infection of the dog or of man, being due to the ingestion of immature parasites in the flesh of the sheep, rabbit, or pig, but only to those diseases in which the parasite goes through the whole cycle of its existence in one host.

The parasites of the mammalia are rarely interchangeable amongst the orders. For instance, the sarcoptic acarus of the dog will not live for any length of time on the horse, or cattle, or sheep, or on man; nay, further, they are rarely interchangeable amongst the families of an order, for the sarcopt of the ferret will not live for any length of time on the cat or dog, and even in families so nearly allied as the fox and dog it is a disputed point whether or not the sarcopt of one will thrive on the other.

It is probable that all animals are subject to scabies. The sarcopt has been found on most of them. I myself have taken specimens from the horse, sheep, dog, fox, cat, ferret, rat, and mouse, besides others, not sarcoptic, from all our domestic animals and from a great many of our wild animals and birds. It is true, though there are cases on record where the disease has been passed on to the point of infecting a cat,

by binding several of the parasites from the skin of one animal to the healthy skin of another animal of a different order or family, and also some other cases where the disease has been contracted by simple cohabitation of animals of different orders, I contend that the former class of cases are no proof, and that the latter are of the rarest occurrence, and further, that when the parasites of one order or family of animals have been introduced on to the skin of an animal of a different order or family in either of these ways, they have not established the disease in that animal, but, after causing a certain amount of irritation, of a more or less transitory nature, the symptoms have abated, and the parasites have died—very different indeed to the persistent, intractable, in many instances, almost incurable, and occasionally fatal cases that are met with when the normal parasite is well established on its normal host.

There are exceptions to this rule, as when, for example, the larva of the *hypoderma bovis* is accidentally introduced under the skin of the horse. The poison of the foreign parasite sets up very aggravated symptoms in its artificial host, but it is, nevertheless, a general rule.

Far be it from me to disparage the value of inoculation experiments, for this is the only reliable method at our disposal to prove whether or not certain organisms, if introduced into the body of a host, will give rise to certain symptoms, and induce certain lesions that are caused by no other organisms, and which cannot be generated by any other means, and if they do so react they must, therefore, be the cause of certain diseases; but I must contend that, except on the very rarest occasions, as when the organisms come in direct contact with wounds either of the skin or mucous membranes, they do not naturally find an ingress in this artificial manner.

I may, perhaps, mention here a very simple experiment of my own as proving the non-reliability of artificial experiment as compared with what really happens in nature.

On the 1st February last I took four apples of a hardy, keeping variety; three of them were quite sound, one was slightly bruised, though the skin was not broken.

In one I made a slight scratch through the skin, and introduced just a speck of the fluid from an apple in which the decaying process had far advanced. Another apple I squeezed hard on to the skin of the decaying one and left them in this position. The third sound one and the bruised one I left in the dark warm cupboard where I kept them all, as being the condition most favourable to induce the propagation of the organisms causing decay or fermentation.

Ten days afterwards a dark soft patch was just visible round the scratch of the inoculated apple—the others were all sound. The dark patch increased fairly rapidly; on the 1st March rather more than one-third of the apple was decayed, and by the middle of the month the whole apple was decayed.

Number 2 sound one that had been in the cupboard, but not in contact, was still quite sound on the 1st March, when I introduced a speck of juice from the decaying apple under a scratch in the skin. Ten days afterwards, decay commenced in the same manner from the spot inoculated, as in my Number 1 experimental apple.

The bruised apple and the one in close contact with the decayed one still were sound, and this last was actually resting in the juice from

the decayed one in a small saucer in which I kept them. On the 20th March, I noticed that the skin had given slightly where the apple had been resting in the juice from the decayed one, and from this point it began to decay. The bruised one, still sound, I now inoculated, and it began to decay, not from the bruise, but from the point of inoculation, a week afterwards.

These experiments may be regarded as simple, childish may be, and they were certainly not very expensive, but they were made to prove the fallacy, or otherwise, of exclusive reliance on laboratory experiments as proving the communicability, or otherwise, of parasitic diseases. It is scarcely necessary to enlarge on the lesson to be learnt from this simple experiment, but one thing is plain, a sound and healthy organism, with, in the case of such an animal as those with which we have to do, a sound skin and mucous membrane, is able to resist decay, or disease-inducing parasites, where an inoculated one becomes a ready prey to them.

As with scabies, so with the pediculi, those of the birds will not live on mammals, and those normally living on mammals are not interchangeable amongst the orders. Those of the carnivora will not live on the bovidæ or equidæ, and *vice-versâ*.

I am mentioning these diseases in order to emphasise the fact that the parasites causing scabies and phthiriasis are creatures of a highly complex organisation, probably quite as much so as man himself, and yet there are very few, even amongst experts, who would be able to pronounce with certainty at sight, if some of these parasites were submitted to them for identification, from what animal they were taken, take, for example, some of the trichodectes, or the acarus scabei, or the symbiotes. But if there is this uncertainty in the identification of these complex creatures, how much greater must be the difficulty of recognising the difference in a parasite which is but a minute rod?

For if we carry this reasoning and these analogies to our consideration of the most recently demonstrated parasitic diseases, of which tuberculosis is one, the minute parasite which is the cause of the disease, both in the ox and in man, appears to be identical. We may take some consolation from the thought that most probably the parasite, which we know lives its whole life cycle in one animal, and which causes a certain sequence of symptoms in that animal, is not transferable, normally, as a disease-producing agency to the body of another animal—not only of a different variety or family, but to man, who is a being of an entirely different order to the ox.

And so it is not enough to say that because certain cows have the bacilli or parasites of tuberculosis in their systems, and certain members of the human race also have them, that therefore the diseases are intercommunicable, and are transmitted normally from the one to the other. To prove that this is, or can be so, we need to be very certain of our proofs and statistics.

Judging from my little experiment with the apples, it would be scarcely right to jump at the conclusion that they may be so transmitted because an animal or a man may become affected, as a result of a certain amount of the virus from one having been artificially introduced into the system of the other. What we have to consider is—would this be likely to occur naturally?

If we come now to a consideration of some of the diseases of an

analogous nature to tuberculosis, *i.e.*, diseases due to the most minute forms of parasites. We can go amongst horses suffering from influenza or strangles with impunity, or amongst dogs with distemper, pigs with swine-fever, cattle with pleuro-pneumonia, and we do not become infected from the contact.

Very rarely indeed does man contract glanders from the horse, and cattle, dogs, and other animals are not susceptible. Horses and men do not contract foot-and-mouth disease from cattle, except perhaps the latter, on the very rarest occasions; for when foot-and-mouth disease was prevalent all through the country there were only one or two recorded cases, and these exceptions prove the rule.

What is the function of these disease-inducing organisms in the rôle of life?

All through the cycle of life it is necessary that there must be a balance; no one form must, for any length of time, exist in superabundant numbers, but other forms keep it in check, except when man comes on the scene and interferes, for a time, with nature's arrangements; but when the top of the scale is reached, there is no higher form to check the superabundant multiplication of man and what we are pleased to term the higher animals, and so the cycle of life is commenced again by the very highest forms becoming the prey of the very lowest.

It is, perhaps, as well to bear this in mind, because it is one of nature's inviolable, unchangeable laws, that she abhors a monstrosity or a weakling; on these she is hard and unmerciful. In the lower forms of life, those that fail most to respond to their environments are those that fall the easiest prey to those next highest in the scale—and so with the highest of all, the physically healthy and vigorous are those best able to resist. With man it is those who keep their bodies in temperance, soberness, and chastity.

And with our animals of domestication, it is those begotten and reared in nearest compliance with nature's laws that are best fitted to resist these ubiquitous foes. The leucocytes of a healthy and vigorous animal or man can, and do, war against them successfully. This being so, it behoves us to see that our stock are kept healthy and disease-resisting, I mean from nature's standpoint.

Whether or no it is ever proved satisfactorily that tuberculosis is naturally transmissible from bovines to man, it is now generally acknowledged that it is so from bovine to bovine. The task we have to face is not only how to stamp it out when we find it existent in a herd, but how to lessen the susceptibility of animals to attack.

On this head attention must be drawn to two very grave errors in our system of stock-rearing.

1st. The stupid, unnatural, though now almost universal, fashion of breeding from immature parents must be a primary cause of the production of stock that become physically unable to resist these bacilli.

Our object should be to tackle this enemy at his base of supplies, and in such a way that he shall have the least chance of getting a hold of our stock at all. We will not wait for him to land, and then devote our energies to kicking him out again, but we will so fortify and guard ourselves that he shall not be able to effect a landing! And how? By creating a breed of stock so vigorous and strong that

they shall be insusceptible of attack ; but this will never be done so long as we countenance the breeding from immature parents. Why, just think of it ! It is the common practice in almost all our great breeding establishments to commence using bulls for breeding at nine months old, or even younger than this ! Aye, and sometimes little female calves, scarcely old enough to be called heifers, are bred from, and this system is carried through several generations.

Is it reasonable to expect the progeny of such parents to be disease resisting ? Of course not ; but it is done, and we wink at it, although we know it is wrong. But then it pays ! Aye, there's the rub, and so it goes on, and so long as stock can be early converted into hard cash, we are not troubled about whether, from nature's standpoint, it is vigorous and strong, and disease resisting, till we find the disease rampant in a herd, when we tackle it, drive it out, and immediately commence again to breed other animals that will, in their turn, assuredly become the victims of these inexorable little defenders of nature's laws, and which she will use in spite of man, to arrest the growth and reproduction of these miserable monstrosities.

If to this first cause is added the second, viz., the unnatural method of the upbringing of much of this stock, there is little need for wonder at the prevalence of this disease in our herds.

In a great many breeding establishments, both gentlemen's and farmers', a rule is that calves and weanlings are kept huddled together in close pens, in closed houses, all through the winter, often in an aspect where they do not get a ray of sunlight, and in semi-darkness. They get no exercise whatever, and they are never cleaned out ; the manure is left to accumulate under them all through the winter. When offering remonstrances, I have been told that they thrive better in this way ; that the heat of the dung keeps them warm ; that if they were turned out they would take cold, etc. Is it any wonder that nature intervenes to check the numbers of such artificial weaklings ?

Our show-rings and the rage for the early maturity of stock are responsible for a great deal of this.

It is our duty as a profession to point out to the stock-breeding public the danger attaching to such breeding and up-bringing, because they are artificially producing a race of animals ever ready to respond to the attacks of these minute organisms.

We can produce a potato that will resist the attack of the *phytophthora infestans*, though infested stocks are growing side by side with it, an example of which I have had in my own garden. And why not, with judicious breeding, healthy surroundings, airy, light, and warm houses, in a good state of sanitation, and good food, produce and keep a disease-resisting stock of cattle ?

These later remarks are not intended to refer to disease generally, though doubtless, if carried into effect, they would tend to protect stock from many diseases (and amongst them probably contagious abortion), but to tuberculosis in particular. For I think we are justified in making two divisions, or classes, of the contagious diseases ; they are all due to parasitic influence, but some of these attack the robust and strong, others only the weakly or neglected, so that we might say some parasitic diseases, such, for example, as pleuro-pneumonia, foot-and-mouth disease, or anthrax are the result of an invasion irrespective of predisposing influences. Others, such

as glanders and tuberculosis, are not the result only but also the cause of an invasion, *i.e.*, in the latter, from a weakly predisposition and from bad sanitation, the vital forces are so lowered as to render the animal an easy prey to the parasites which nature is using for the destruction of the beings that do not conform to their environments. In the other case the parasites are able to attack weak and strong alike.

Let us go again to nature's laboratory for an illustration to show why tuberculosis may be regarded as a cause of invasion, or the effect of a weakly constitution, for I believe it only attacks successfully the weakly, or what we are pleased to call those predisposed by heredity. I take my analogies again from observations in my country rounds. I could point out oak and elm trees that I have been interestedly watching for some few years, gradually being killed and returned to the elements by the larvæ of the goat-moth, which are year by year taking to themselves the vital sap, and destroying the vital energies of the trees, almost exactly as we see consumptive patients gradually wasting away. But these trees have in every case been first weakened by interference with their roots, by the making of banks or ditches, so that the lowering of the vital energies is in these cases the cause of the invasion of the larvæ.

From observations amongst cattle and men it appears to me that this is the law of attack by tubercle bacilli; those whose health and vigour have been sapped from some cause or other. In-and-in breeding, immature parents, unhealthy surroundings (more particularly in early life), hereditary predisposition, or vital functions lowered by exposure, hardship, over-work, or disease, are the conditions that render individuals most susceptible of attack. We have another analogy in the case of pediculi; it may be noticed frequently in cart-horse stables that old and weakly horses are badly affected, and young, hardy ones standing next to them escape.

On the other hand, the larvæ of some insects attack the foliage of the strongest growing trees—oaks, pines, etc.—these are nature's pruners, and these attacks appear to me to be analogous to those parasitic diseases of animals, where the robust and strong are the victims; the rôle of the parasites in both instances being to keep in check over luxuriant growth in the individual, or in families or orders of creatures.

Might the suggestion here be made that it would enhance the interest of inoculation experiments if it could be ascertained whether the animals used for experimental purposes were the produce of mature or immature parents, for, say a generation or two, and whether they were reared under fairly sanitary conditions?

A word or two on the duties and responsibilities of the veterinary surgeon in dealing with tuberculosis in a herd of cattle.

The introduction of tuberculin into the system will, by certain recognised results, give a fairly precise indication whether the animal infected has or has not the parasitic bacilli of tuberculosis in the system; and it seems at first sight a legitimate, fair, and proper thing for any owner of cattle to apply the test to his stock to ascertain which are healthy and which are diseased, and it would be quite the correct thing to do if all reacting animals were placed under the supervision and control of a properly constituted authority; otherwise

it is a delusion, a snare, and a sham, for there is nothing to prevent a man putting these reacting animals on the market to victimise an unsuspecting public.

I take it that this is one of the reasons why tuberculin is only supplied to veterinary surgeons, because it is assumed that they would not lend themselves to be the aiders or abettors in such proceedings, and yet, unless they have a guarantee of what is to be done with reacting animals, they are placed in this position.

I was very much struck with the remarks of a gentleman during a discussion on this subject on another occasion. He said that he considered it was no business of the veterinary surgeon what became of these animals after the application of the test. He performed certain duties for which he was paid, and there his responsibility ceased.

I fail to see it in that light, but if this is the correct interpretation of our position why should the use of tuberculin as a test be restricted to us only? Why not let any stock-owner have it to test his own cows, and do with them afterwards as he thinks best? The reason must surely be that we are regarded as the "buffer" between stock-owners and the public, to protect the latter from any injustice that might so easily be practised by an improper use of tuberculin.

But, again, we should not regard tuberculin, neither should the public be led to look upon it in the light of the sovereign remedy and the sovereign preventative of tuberculosis. We may argue that, by its use, we may eliminate tuberculosis from a herd, and then, by applying the test to all new purchases, and by rejecting those that react, we may prevent its re-introduction.

But what is the ordinary dairy-farmer, on whose behalf we are supposed now to be acting, going to do with those that react and with his new purchases that will not stand the test? Will he slaughter them and bury them? or will he send them back to market for someone else? Which is the most likely procedure for the average dealer in cattle? He is no worse than other men, still he is human, and by re-selling he is breaking no law (except the moral law, which does not count), but only falling back on an unorthodox law of the unregenerate, but still a law that is in evidence in all parts of the universe, from the illimitable mighty systems of the heavens to the ultimate elements of the most inconceivable smallest speck of matter—"Action and reaction are equal and opposite."

But we have to go further than merely detecting the disease and preventing its re-introduction by the tuberculin test; it is for us to insist on the conditions I have already mentioned—the breeding, upbringing, sanitary conditions, etc., of the young stock—to make them proof against attack.

When the bacilli of tuberculosis are found in the absorbent glands only, it is probable, generally speaking, that the animal only so affected is, for the time, harmless. For, unless a vital organ is affected, it is doubtful whether the host suffers from the invasion, at least so long as its vital functions are vigorous, for such an animal will fatten and thrive and not show clinical symptoms of disease, which could scarcely be the case if any of the vital functions were interfered with.

In conclusion, I would say that there can, and may be occasionally, the rare infection of man from bovines, but that it is not nearly so common or easy an occurrence as has lately been generally supposed; but even if it is only possible, we should be careful to eliminate, as far as possible, the cause of and tendency to tuberculosis from our cattle, and also take every precaution to prevent the conveyance of the bacilli from cattle to man. For even if it is only of rare occurrence that man may contract the disease in this way, what a terrible, cruel, unrelenting disease it is! And the being so attacked might be the child or dearest friend of the reader, or of the writer of this article. This, though savouring of the selfish, is the best way to bring the seriousness of the question home to our individual selves.

I don't think any of us would like the idea of our children being given milk from a known tuberculous cow, neither should we care, knowingly, to be eating tuberculous meat, and if we would not like it ourselves it is surely our duty to see that our less privileged neighbours have not these products forced upon them.

But here again, probably, our part-knowledge makes us more timorous than we should be if we knew the whole truth, for the knowledge of the existence of parasites on any being creates a very natural feeling of repugnance, and a dread that they might be conveyed from animals to man. Even medical practitioners are not altogether free from participation in this dread, and it is only a knowledge of biology and the laws of life that enables us to speak confidently of the inconclusiveness of their deductions.

Ladies will tell you, with horror, that their pet dogs are infested with those horrid, unnameable creatures! Is there any risk of their "taking them"?

Ask a man, who is ignorant of the matter, to pluck a fowl, and he will come to you a very little time afterwards in a state of great perturbation, saying he has "caught the lice" from the fowl.

We can tell them, in both instances, that their fears are groundless, that these parasites of the dog or fowl will not live on man (or woman). And it may be, in the near future, that we may be able to speak with the same confidence with regard to these infinitely smaller vegetable parasites, when the first flush of the excitement at the discovery of the parasite itself shall have cooled down.

SOME EXPERIMENTS ON THE TEMPERATURE NECESSARY FOR KILLING TUBERCLE BACILLI IN MILK.¹

By PROFESSOR BANG, Copenhagen.

THE question of the thermal death-point of tubercle bacilli seems not to be solved; at least you will find different opinions stated from well esteemed authors of the latest years. I therefore hope that some experiments shortly made by me and my assistant, Mr. Stribolt, regarding the temperature necessary to kill tubercle bacilli in milk, will have some interest.

Our experiments regard milk secreted from a tuberculous udder; the appearance of the milk was not much altered, a little thinner than natural and flocculent, so that the results obtained refer to the natural conditions in which tubercle bacilli are met with in milk.

In one series of experiments we treated the milk in a quantity of 25 cc. in large test tubes, which we put into a water bath of a constant temperature of 90° C. In the midst of the milk was applied a thermometer fixed in a cork, and as soon as the thermometer showed the degree of temperature which we wanted the tube was removed and cooled in ice water. It took from 1 min. 20 secs. to 2 mins. 50 secs. to reach the various temperatures from the initial temperature of 20°.

Of the heated milk we injected 10 cc. into the peritoneal cavity of rabbits. The result was that eighteen rabbits which received the milk heated to 75° (4 rabbits) — 80° (4), 85° (6), 90° (4) remained healthy (or died after some months of other diseases), while two rabbits injected with the raw milk died after about two months highly tuberculous.

In this series of experiments we could not avoid the formation of a thin pellicle on the surface of the milk, which, after the experience of Professor Theobald Smith, is supposed to protect the bacilli included therein against the influence of the heat; nor could we avoid the formation on the surface of a layer of foam, which might possibly have the same effect. And we found, on the other hand, that the temperature of the milk was not equal through the whole mass of milk—it was, indeed, about 5° higher near the surface than near the bottom—when we did not shake the tube, which we wanted to avoid because we feared that particles of the milk hanging on the thermometer might be protected against the heat.

In order to avoid all these possible sources of error and to secure a thorough heating of all particles of the milk, we altered our method of heating, and included the sample of milk in a thin metallic box of cylindrical form, closed with a metallic cover, in which the thermometer was fixed. The box contained 30 cc. of milk, and when we put the whole deeply into a large water bath of constant

¹ Communicated to the British Congress on Tuberculosis, July 1901.

temperature, shaking it the whole time, we were sure that no particle of the milk could avoid the influence of the heat. In such a metal box the milk very soon, after about 1 minute, reaches the temperature of the surrounding water. In some cases we cooled the milk in ice water as soon as the temperature wanted was reached, in others we kept it at this temperature 1, 2, 5, 10, 15 mins.

Of tuberculous milk treated thus we injected into the peritoneal cavity of rabbits 10-15 cc.

The result will be seen by the following table:—

<i>Date of Injection.</i>	<i>Quantity Injected.</i>	<i>Temp. of the Milk.</i>	<i>Time of Exposure.</i>	<i>Rabbits</i>	<i>Date when the animal died or was killed.</i>	<i>Result.</i>
March 23	15 cc.	85° C.	Momentary	1	Killed July 8	Sound.
—	—	—	1 min.	1	Killed July 8	Sound.
—	—	80° C.	Mom.	1	Killed July 16	Sound.
—	—	—	1 min.	1	Killed June 6	Sound.
—	—	75° C.	Mom.	1	Killed July 16	Sound.
—	—	—	1 min.	1	Killed June 6	Sound.
—	—	70° C.	Mom.	1	Killed May 4	Sound.
—	—	—	1 min.	1	Killed June 4	No tuberculosis. Pneumonia.
—	—	65° C.	Mom.	1	Killed May 4	Tuberculosis in moderate degree. The rabbit was rather fat.
—	—	—	1 min.	1	Died April 2	No manifest pathological alterations.
—	10 cc.	—	5 mins.	2	Killed May 4	No tuberculosis. One of them had a large cheesy deposit (not tuberculosis) in the peritoneal cavity.
—	—	—	10 mins.	2	(a) Died Mar. 20	No tuberculosis. (Peritonitis).
—	—	—	15 mins.	2	(b) Killed Mar. 20	Sound.
—	15 cc.	60° C.	Mom.	1	Killed June 10	Both sound.
—	—	—	1 min.	1	Died May 2	Highly tuberculous.
—	—	—	—	1	Killed May 4	Highly tuberculous.
—	—	—	—	1	(a) Died April 1	Peritonitis. No tubercle bacilli to be found.
March 24	10 cc.	—	5 mins.	4	(b) Killed May 4	Tuberculous, rather highly.
—	—	—	—	4	(c) Killed May 2	Tuberculous, rather highly.
—	—	—	—	4	(d) Killed May 12	Tuberculous, rather highly.
—	—	—	—	4	(a & b) K. May 12	Both slightly tuberculous.
—	—	—	10 mins.	4	(c) Died April 15	No tuberculosis. (Pneumonia).
—	—	—	—	4	(d) Killed June 10	Tuberculous, rather highly.
—	—	—	—	4	(a) Killed May 21	Very few tubercles in the omentum, a mesenteric gland, and the lungs.
—	—	—	15 mins.	4	(b) Killed May 21	Sound.
—	—	—	—	4	(c) Died May 25	Very slightly tuberculous (as a). Died of broncho-pneumonia.
—	—	—	—	4	(d) Killed June 6	Sound.

FEEDING EXPERIMENTS.

With the same tuberculous milk we fed, 17th March, a number of young rabbits. Each rabbit got 30 cc. of the milk heated in the above-mentioned way in the metallic box and kept two minutes at the temperature whose influence we wished to examine.

The result will be seen on the following table :—

<i>Date of Feeding.</i>	<i>Quantity given.</i>	<i>Temp.</i>	<i>Animals.</i>	<i>Date when the animal died or was killed.</i>	<i>Result.</i>
March 17	30 cc.	85° C.	2 rabbits	Killed July 8 . . .	Both sound.
—	—	80° C.	2 rabbits	{ (a) Died April 20 . . . (b) Killed July 8 . . .	No tubercle. Sound.
—	—	75° C.	2 rabbits	{ (a) Died June 7 . . . (b) Killed July 8 . . .	No tubercle. Sound.
—	—	70° C.	4 rabbits	{ (a) Died March 28 . . . (b) Died May 24 . . . (c & d) Killed July 8 . . .	No tubercle. (Was bitten to death). No tubercle. Both sound.
—	—	65° C.	2 rabbits	Killed June 16 . . .	Both sound.
—	—	60° C.	2 rabbits	Killed June 6 . . .	Both sound.
—	—	Raw milk	2 rabbits	Killed June 2 . . .	Both <i>tuberculous ulcers</i> in the intestines (on the Peyer's patch in the ileum and in the end of cæcum). <i>Tuberculosis</i> in the mesenteric glands, in the liver, spleen, kidneys, and lungs.

It will be seen that although the heating of tuberculous milk to 60° c. for fifteen minutes is not able to destroy all tubercle bacilli, so as to prohibit infection if you inject them into the peritoneal cavity, the heating to the same degree for two minutes is enough to weaken them so much that they cannot infect through the alimentary canal.

We also made some feeding experiments with pigs. Each pig got 150 cc. of the same tuberculous milk, heated in the metallic box, and kept half a minute at the temperature whose influence we wished to examine.

The result was the following :—

<i>Date of Feeding.</i>	<i>Quantity given.</i>	<i>Temp.</i>	<i>Animals.</i>	<i>Date when the pig was killed.</i>	<i>Result.</i>
March 21	150 cc.	80° C.	1 pig	June 16 . . .	Sound.
—	—	75° C.	1 pig	July 1 . . .	Sound.
—	—	70° C.	2 pigs	July 9 . . .	Both sound.
—	—	60° C.	1 pig	June 13 . . .	<i>Tuberculosis</i> in the lymphatic glands near the throat, in the mesenteric, mediastinal, and bronchial glands, and some nodules in the lungs.
—	—	Raw milk.	1 pig	June 13 . . .	Sound.

This experiment does not, of course, prove much regarding the influence of the heat, as the pig that ate the raw milk did not acquire tuberculosis. As Mr Stribolt himself saw it drink the tuberculous milk, the experiment has some interest, inasmuch as it proves that a pig may resist one feeding with a small quantity of tuberculous milk.

As to the influence which the quantity consumed has on the development of the disease, I wish to mention here some experiments with calves which we made two years ago. 4th March we gave each of five sound calves (three and a half months old) 40 cc. of milk from a tuberculous udder, and six days later they got the same dose. When these calves were killed after three to nine months we found them all tuberculous with deposits in the mesenteric glands, some also in the supra-pharyngeal glands, and some with tuberculous ulceration in the intestines. One of them was much emaciated and had profuse diarrhoea. It had enormous tuberculosis in the bowels and the mesenteric and supra-pharyngeal glands, and also some tuberculosis of the lungs and bronchial glands.¹

It is thus proved that calves may sometimes become tuberculous after feeding with a very small quantity of tuberculous milk.

EXPERIMENTS WITH TUBERCLE BACILLI IN CULTURE.

In order to complete our researches on the influence of the temperature on tubercle bacilli, we made some experiments with pure cultures. In small test tubes with glycerine-bouillon we sowed small particles of the thin pellicle which a young culture of tubercle bacilli forms. The cotton-wool plug was pushed downward and a caoutchouc cork (perforated by a small glass tube with cotton-wool) put over it.

These test tubes we put deeply down into a water bath in order to acquire as much security as possible for the thorough heating of the thin pellicle of culture which swam on the surface of the bouillon. We knew from experience that it takes about $1\frac{1}{2}$ minutes before the bouillon has the same temperature as the surrounding water, and therefore we no doubt are right in speaking of a momentary heating to a certain degree when we removed the test tube from the water bath after $1\frac{1}{2}$ minutes and put it into cold water. Accordingly we speak of 5 minutes' heating when we removed the test tube after $6\frac{1}{2}$ minutes, and so on.

The result of our experiments was that the culture grew as well as in the control tubes after exposure to a temperature of 50° C. for 15 minutes; a little more slowly after 50° C. for 20 minutes.

55° C. had no influence by momentary action; after 5 minutes the growth was retarded, after 10 minutes some tubes got sterile, and after 15 minutes only one single tube showed a trace of growth.

60° C. made most tubes sterile after momentary action; in a few tubes there came a slow growth.

60° C. for 5 minutes made all tubes sterile.

65° C. made most tubes sterile after momentary action; in some tubes there was a doubtful trace of growth.

¹ Three of these calves were before the feeding (and one of them also after it) treated with large quantities (128 cc. each) of the new tuberculin (T.R.), but it had no influence at all. The most affected was one of the treated.

65° C. for 5 minutes made all tubes sterile; and the same was the case when the temperature had acted for a longer time, and when we exposed the tubes to higher temperatures as 70°, 75°, 80°, and 85°—in all cases no trace of growth.

These experiments with pure cultures of tubercle bacilli correspond then quite well with the milk experiments; the influence of the heat is only a little stronger on the cultures. Maybe the cultivated bacilli are a little weakened, maybe they would be able to grow in the peritoneal cavity of an animal even after having lost the power of multiplying in bouillon with glycerine.

Our experiments have given almost the same results as those published by Professor Theobald Smith two years ago (*Journal of Experimental Medicine*, 1899). He found tubercle bacilli destroyed at 60° C in 15 to 20 minutes—and the larger number in 5 to 10 minutes, but they differ from what has been found by many other researchers, and also by myself many years ago. I occupied myself in 1884 and the following years much with experiments with milk from tuberculous udders, and I came to the result that, although the heating to 65° C for 5 minutes in some cases made the milk innocuous, this was not always the case. The same inconstancy I found for the higher temperatures, as 70°, 75°, and 80°. But 85° C I constantly found able to kill tubercle bacilli in milk. By feeding experiments in rabbits I demonstrated the weakening influence at 60° C and 65° C, in so far that milk heated to these temperatures could only produce very slight tuberculosis in some individuals, and in some cases was quite innocuous, although the feeding was continued for three months. At that time I felt inclined to seek the reason of the inconstancy of the result by injection in variability of the tubercle bacilli (perhaps spores), but nowadays I think it more probable that it depended upon faults in the method of treating the milk. As that was done in open test tubes put into a water bath, the formation of a pellicle on the surface was not avoided, nor the formation of a layer of foam on the surface. And as no doubt the test tube was shaken a little, particles of milk may have been deposited on the thermometer above the surface of the milk, and in this way have been protected against the heat. Then the inconstancy may in some cases have depended upon the condition of the milk, which in some cases was rather serous and disposed to coagulation, and in some cases also contained rather large flakes, in whose interior the bacilli may have been somewhat protected.

I suppose that some of these circumstances may explain the fact that many other investigators have had results which also differ from those of Mr Theobald Smith, and Mr Stribolt's and mine.

Most corresponding to our results are Dr De Mann's, of the Netherlands (*Archiv für Hygiene*, 1893). He found the tubercle bacilli killed after 15 minutes' exposure to 65° C, after 10 minutes to 70° C, and after 5 minutes to 80° C. But he relates no experiments about the influence of these temperatures applied for a shorter time, and his researches consequently do not enlighten the thermal deathpoint of tubercle bacilli.

The same thing may be said of the researches of Yersin (*Annales de l'Institut Pasteur*, 1888), who found that 70° C. in 10 minutes was able to kill the bacilli, but he did not try if 70° C. could have done

the same thing in a shorter time. Nor did he try the influence of 65° C., but for 60° C. in 10 minutes he found a weakening influence.

More different from my results are those of Prof. Max Beck (*Vierteljahrsschrift für off. Gesundh. pflege* 1900). He found that simple boiling was not sufficient to kill the bacilli in natural tuberculous milk, and that milk mixed with tubercle bacilli was not made innocuous by heating to 80° C. for half an hour, but his experiments seem to have been done by heating the milk in open vessels, and it is then permissible to believe that the formation of a pellicle or of foam on the surface has been the reason of the deviating results.

In the large series of researches made by Prof. Woodhead in 1895 for the Royal Commission of Tuberculosis, it will be seen that as long as he worked with natural tuberculous milk most of his results correspond very well with mine, inasmuch as milk treated to 60° C. for 5 minutes did not produce tuberculosis in guinea-pigs with feeding, and such milk heated to 60° C. for 25 minutes did not make them tuberculous after injection into the peritoneal cavity, and the same was the case with milk heated to 65° C. for 2½ minutes. Only in one experiment he found that after heating to 70° C. for 5 minutes the milk made one out of three guinea-pigs tuberculous by injection, and the same result he got with two guinea-pigs injected with milk heated to 70° C. for 11-12 minutes, while five guinea-pigs which got the milk heated for 9-10 minutes remained free.

With milk mixed with finely divided tuberculous organs Prof. Woodhead had very inconstant results, but I think it is permissible to believe that in these cases some tubercle bacilli had been protected against the heat through coagulation—a circumstance to which he himself calls attention.

Finally, I wish to point out that it would no doubt be wrong immediately to draw consequences for practice from these scientific researches. In a common pasteurising apparatus for milk, as it is used in the dairies, the milk passes so fast (in some of them in a little more than half a minute) that it does not quite correspond to what I call momentary treatment in our metallic box, and in most of them the milk is very foamy, and we have found that the foam which comes out of such apparatus is not at all as hot as the milk. Then the milk may in some cases contain large flocculi of coagulated material. It would therefore I think be unlucky to diminish the demand as to the degree of heating in the common dairies. I think it best to continue the heating of the skimmed milk before it is sent back to 80° or 85° C., as we do in Denmark. Only at this temperature is it possible to control the heating by a simple colour-test (method of Storch, *i.e.*, shaking of the milk with some drops of paraphenylen diamine and hydrogen dioxide).



HOW CAN THE TUBERCULIN TEST BE UTILISED FOR THE STAMPING OUT OF BOVINE TUBERCULOSIS.¹

By SHERIDAN DELÉPINE, M.B., Edin., B.Sc., Lausanne,
Proctor Professor of Pathology, Owens College, Manchester.

PART I.—DIAGNOSIS.

A. Preliminary Remarks.—The question before us is the stamping out of bovine tuberculosis, and more especially how the use of tuberculin can assist us in obtaining this result. The prominence given to tuberculin in this discussion is due to the fact that in our estimation, as well as in that of the most competent authorities, Koch's great discovery gives us the most efficient means of diagnosing tuberculosis during life. In my estimation the keystone of preventive measures must be a reliable and readily applicable method of diagnosis. I might be excused if, after stating this view, supported as it is by the weight of the highest authorities, I made no further attempt to prove its correctness. By following such a course my right to express a definite opinion might be challenged, and I might offer further food for the criticism of those who are still supporting our responsible governments in their attitude of agnosticism. Such an attitude is well expressed by Mr Walter Long, the late President of the Board of Agriculture, in his 1899 Newcastle speech, in which he is reported to have said :—

1. The data were still too indefinite and wanting in preciseness to justify them in asking Parliament for great votes of public money, or to impose on stock-keepers compulsory measures entailing great loss. 2. They were not yet sure that those measures would be effectual. 3. The tuberculin test might be administered so as to render cattle incapable of reaction on a second application of the test, and that might be productive of fraud. 4. People who had studied the question most closely were not agreed as to the conditions under which the test should be applied. 5. The disease was difficult of detection unless one could rely on some such discovery as the tuberculin test. 6. At present too little was known, too much doubted, for Parliament to be justified in imposing upon the country heavy expenditure on wholesale restrictions which would be strongly resisted in many quarters, and which might not do anything effectual for the extinction of the disease.² Mr Long's contentions receive support from Schütz's observations.³ In 1891 this observer tested 66 head of cattle—51 reacted, of these 43 only were tuberculous; 15 did not react, and of these, four were tuberculous. They are also supported by statements made by Professor Bang at the Congrès pour l'Étude de la Tuberculose chez l'Homme et chez les Animaux, held at Paris in 1898.⁴ Mr Walter Long's criticism, which was made some time after the publication of Professor Bang's communication, seems at first sight to express identical views. A careful consideration of Professor Bang's state-

¹ Abstract of a paper communicated to the British Congress on Tuberculosis. Section I., State and Municipal. Reprinted from the *Lancet*.

² "Manchester Guardian," 30th Jan. 1899.

³ Schütz: "Arbeiten aus dem Kaiserlichen Gesundheitsamte," Band VIII. See also Nocard: "Annales de l'Institut Pasteur," 1892, VI., p. 50.

⁴ "La Semaine Médicale," 1898, p. 329.

ments will, however, show that they do not entirely justify Mr Long's conclusions. Professor Bang emphasises the reliability of positive reactions in the great majority of cases; he points out the conditions which may bring about failure of reaction, and also the ways by which such errors may be avoided or considerably reduced. He shows also that most of the tuberculous cases which do not react are, firstly, cases in an advanced state of tuberculosis, *i.e.*, those which can be easily diagnosed by other means; secondly, cases which have previously reacted and have acquired tolerance; and thirdly, cases in which a slight tuberculosis has been present, but in which the disease is becoming arrested. I will be able to show that such cases should be no obstacle to proper administration. I wish also to emphasise the fact that although Professor Bang's restrictions may have been used by others for purposes of adverse criticism their author is not responsible for this, and is, undoubtedly, one of the earliest and strongest supporters of the use of tuberculin in diagnosis.

We have, on the other hand, statements made by equally reliable and recognised authorities, which are entirely favourable to tuberculin. I will not lengthen this communication by a detailed account of all the records available, for my purpose will be equally well served by quoting the conclusions which have been arrived at by Professor Nocard, and by the Royal Commission on Tuberculosis, after a careful compilation of records available and the carrying out of confirmatory experiments. Professor Nocard in 1892¹ gave a very useful summary of the earlier work done soon after the announcement of Koch's discovery of tuberculin, which he summed up as follows:—
 "Tout le monde admet aujourd'hui l'exactitude et la précision merveilleuse des indications diagnostiques fournies par la tuberculine."

After quoting Professor M'Fadyean's opinion and his "most implicit faith in tuberculin as a test of tuberculosis when it is used on animals standing in their own premises and undisturbed," the Royal Commissioners in their report on tuberculosis state: "We entirely accept these conclusions and entertain no doubt as to the value of tuberculin, provided the test is applied by a competent veterinary surgeon and that the tuberculin is of trustworthy quality."²

B. Criticism of previous Experiments regarding the Value of Tuberculin.—It is not difficult to account for the differences of opinion which have arisen out of the results of early experiments with tuberculin.

1. The reaction is not simply manifested by any rise of temperature, but, as Nocard and others have frequently observed, this rise of temperature is characteristic only under certain conditions which must be observed in order that the results should be reliable. (*a*) The animal submitted to the test should not be at the time subject to sudden variations of temperature, or be suffering from pyrexia. Before applying the test it is therefore necessary to observe the animal for two or three days, and to take its temperature morning and evening. Recently driven cattle especially should be tested only after they have been submitted to this preliminary observation. The calving period is also to be avoided. (*b*) In tuberculous animals a characteristic rise of temperature is usually observed between the

¹ "Annales de l'Institut Pasteur," Vol. VII., p. 44.

² "Report of the Royal Commission on Tuberculosis," Part I., Art. 13, p. 4, 1898.

ninth and fifteenth hours, but it is occasionally, though rarely, observed after the fifteenth hour. (c) A slight rise of temperature not amounting to more than 1° C. above the normal temperature of the animal, cannot be considered characteristic, since healthy animals are liable to fluctuations of temperature capable of producing such a rise. It is therefore desirable to adopt some scale similar to that recommended by Nocard who considers a rise of 0.8° C. (1.4° F.) as having no significance; a rise of from 0.8° to 1.4° C. (from 1.4° to 2.5° F.) as being only suspicious and requiring the confirmation of a further test at the end of a month; a rise of from 1.5° to 3° C. (from 2.5° to 5.4° F.) as characteristic of tuberculosis when the conditions previously stated have been observed. (d) It is almost useless to add that no tuberculin should be used which does not come from a reliable source and which has been exposed to loss of activity by long keeping. The exact dose of each preparation should be carefully ascertained. Many of the earlier observers have not paid sufficient attention to these precautions. Thus Schütz has considered a rise of 0.5° C. as indicating a tuberculous reaction.

2. Sufficient care has not always been taken to make a thorough *post-mortem* search for minute tuberculous lesions on animals which had reacted, and in the absence of very careful and minute examination it is unscientific to report an entire absence of tuberculosis.

3. The absence of reaction in animals suffering from advanced tuberculosis does not affect the value of the test. This absence of reaction is only what can be expected in the case of animals which are already saturated with the products of the tubercle bacillus. This state is probably analogous to the state of tolerance produced by frequently repeated doses of tuberculin. But when cows have reached such a stage of tuberculosis their state should be obvious to a competent veterinary surgeon, and as most of these animals would be suffering from advanced phthisis the clinical diagnosis could be confirmed by the bacteriological examination of mucus, obtained by swabbing the pharynx according to the method recommended by Greffier.¹ (To excite secretion, pilocarpine, from 25 to 30 centigrammes may be previously injected.)

4. The absence of reaction in the case of animals in an early stage of tuberculosis will, I think, be found to be very rare when the test is properly applied. The most remarkable feature of the tuberculin test is the power which it gives to detect extraordinarily small lesions, and it is this property which, I think, renders it so specially suitable as a basis for preventive methods, and will allow such methods to be carried out economically, *i.e.*, before infected animals have lost their value on the meat-market. But even if a few escaped it is obvious that early tuberculous lesions could hardly be a source of infection in other animals, and that under a regular and thorough system of inspection these animals would be detected before they had become a source of danger to other animals or to man.

5. With regard to the tolerance acquired by cattle which have been tested several times, I can best indicate to what an extent this might be a source of difficulty, by giving the results of some experiments made for the Cheshire County Council by Mr Laithwood, chief veterinary inspector of the county. I was able to follow these experi-

¹ "Bulletin de la Société Centrale de Médecine et Vétérinaire," 1890, p. 445.

ments closely, being engaged in another branch of the same investigation. In the space of eighty days several cows were tested five times at variable intervals with the following results (Table I.).

TABLE I.—*Effects of Testing with Tuberculin Repeated at Short Intervals.*

Reference No. of Cow.	First testing.	Second testing, 45 days after first.	Third testing, 13 days after second.	Fourth testing, 14 days after third.	Fifth testing, 12 days after fourth.	Result of post-mortem examination and remarks.
5	?	?	—	?	—	Case of distomatosis ; temperature irregular.
24	+	+	+	+	—	Advanced tuberculosis.
32	+	+	+	+	?	Slight tuberculosis.
37	+	+	+	+	—	Slight tuberculosis.
41	+	+	+	+	—	Advanced tuberculosis.
42	+	+	?	+	—	Advanced tuberculosis.
27	+	+	Not tested	+	Not tested	Advanced tuberculosis.
35	?	—	Not tested	—	Not tested	Healthy.
51	+	+	In calf ; not tested	+ ¹	...	Tuberculosis slight.

Three of the cows which had ceased to react were found to be still in the same state 35 days after the last test. ? indicates that the reaction was doubtful.

It was only at the fifth injection that the tuberculous cows all ceased to give a reaction, and this fifth injection had followed the fourth, third, and second testings at intervals of only 12, 14, and 13 days respectively. Nocard had previously obtained more carefully tested results showing that tolerance to tuberculin is not so easily produced as has been stated.

Fear of fraud cannot be considered a very sound argument against the use of a good method.

C. New Experiments.—Having shortly referred to some of the evidence already available, I must now record the results of experiments which have been conducted under my supervision since 1897, and in which an attempt has been made to observe all the precautions necessary to avoid the fallacies which have been previously discussed. I will also take advantage of the extensive investigation carried out in Aberdeen on behalf of the University Department of Agriculture in 1899. As a result of a conversation which I had with him, Mr Gair Ashton, M.P., in 1897 decided to test practically the possibility of stamping out tuberculosis from the herd kept on his estate at Ford Bank. The probable cost of the experiment which I fully explained did not deter him, and with great liberality and public spirit he gave me the means of obtaining all the information which I could obtain

¹ Tested later than the others.

from such an experiment. The small herd kept at Ford Bank consisted in 1897 of 21 cows, bullocks, heifers, and calves, several of which were tuberculous. The problems before us were, first, to stamp tuberculosis out of Ford Bank; secondly, to estimate the cost of this radical procedure; and, thirdly, to keep the new stock free from tuberculosis. Briefly speaking, these experiments were conducted as follows:—

All the stock on the farm was tested with tuberculin from the Institut Pasteur by Mr Locke, assisted at first by Mr King, the corporation chief veterinary inspector, and afterwards by Mr Holburn, Mr King's successor. The test was applied with all the precautions which I thought necessary, and which I have previously indicated. In all cases the animals were kept under observation for two or three days before testing, and their temperature was taken at least three or four times. After the injection of tuberculin the temperature was taken, with few exceptions, four times between the ninth and twentieth hours, at intervals of three hours. The animals which did not react were at once transferred to recently disinfected sheds. Those which reacted were speedily sent to the abattoir. The carcasses of slaughtered animals were examined carefully at the abattoir by Mr Holburn, and the lesions found were carefully recorded. In doubtful cases the organs or parts of organs were referred to me for further investigation. The price obtained for each carcase was also noted. Each animal previously to being slaughtered was valued by an independent expert. All the calves were kept isolated from the first and were fed on boiled milk. No attempt was made to separate the calves of tuberculous dams from the others, of which there were very few. The stables which had been occupied by tuberculous cattle were disinfected thoroughly with chlorinated lime solution and then they were white-washed. To fill the gaps made in the old stock new stock was bought subject to the tuberculin test, the animals being kept in a separate shed previously to the application of the test. All animals found to react were returned to the seller and only those which did not react were added to the old stock. This was done in order to ascertain the safety of relying on the tuberculin reaction as a basis for contract. The milk of none of the tuberculous cows was again used on the farm without being boiled, although there was no evidence of tuberculosis of the udder in any case. Finally, owing to Mr Ashton's change of residence, all his cattle were sold by auction and a record was kept of the prices obtained for each animal. Shortly speaking, the results of the tests were as follows in the case of cattle which I have been able to keep sufficiently long under observation. (I have excluded all animals which after being tested once were lost sight of.) *Definite reactions*: 48 out of 49, *i.e.*, nearly 98 per cent. The doubtful reaction gave place to a definite reaction on the second trial within three months, so that the failure was not a permanent one. *Positive reactions*: 19 out of 19 proved to be correct, *i.e.*, 100 per cent. *Negative reactions*: 28 out of 29 proved to be correct at once, *i.e.*, 96·5 per cent. The tuberculous cow which did not react had newly calved; this error was corrected within three months by re-testing. I believe that a method which gives such results may be considered as perfect as any other method of diagnosis can ever be expected to be.¹

I am, fortunately, in a position to avail myself of the results of a

¹ See second part of this paper for the other results of this experiment.

later investigation bearing on a much larger number of animals examined under conditions such as may be easily observed in practice. I had opportunity to discuss with Professor D. J. Hamilton the conditions under which this set of experiments was carried out, and I am fully satisfied that, as far as they go, the results may be received as absolutely reliable. I feel, however, that, owing to the facts that the temperature of the animals was taken only once before the test was applied and only between the tenth and thirteenth hours after the injection, the value of tuberculin was tested somewhat more severely than was necessary. It is not possible to reproduce here the tables of results given in the report of this experiment published in 1899 by the Department of Agriculture of the Aberdeen University.¹ The authors explain in this report that by arrangement with important butchers and cattle-owners they have been able to test a large number of cattle which were intended for slaughter, generally on the farm before removal, but when this was impossible the test was applied in the byre connected with the slaughter-house after the animals had been kept there for twenty-four hours. It will be obvious, therefore, that in the second group of cases the test was applied under conditions not altogether favourable to success. The main conclusions of the authors regarding the value of tuberculin are summed up as follows by themselves: "1. Our experience confirms the generally accepted opinion that tuberculin loses its virulency when kept for any length of time. 2. When used with care and under proper conditions tuberculin is a reliable diagnostic of tuberculosis in cattle, except (*a*) when the tubercular lesion is minute, or (*b*) when the disease has become generalised, especially in the case of aged and emaciated animals." The authors sum up these results in four tables, giving a detailed account of the age of the animals tested, of the temperature observed, and of the lesions found in the carcasses after slaughter. I have prepared on the basis of their own records the following table (Table II.).

TABLE II.—*Analysis of the Sources of Failure in the Aberdeen Experiments.*

	Number tested.	Age.	Tuberculin reaction.		Tuberculosis found post-mortem.	Error in tuberculin reaction.	
			—	+		—	+
Heifers . . .	60	1½ to 2 years	54	6	10	Per cent. 0.0	Per cent. 6.6 ²
Bullocks . . .	100	2 years	80	20	24	0.0	5.0 ³
Bulls . . .	3	3 years	2	1	1	0.0	0.0
Cows . . .	77	Average 7½ years	52	25	42	0.0	32.6

¹ "Report on an Investigation with regard to the Value of Tuberculin as a Test of the Presence of Tuberculosis in Cattle." By J. McLauchlan Young, F.R.C.V.S. and J. S. Walker, M.B., D.P.H., Aberdeen University.

² Corrected 1.6 per cent.

³ Corrected 2.5 per cent.

NATURE OF CASES IN WHICH ERROR WAS OBSERVED.

Heifers.—Nos. 28 and 30: bad tuberculin used. No. 31: the syringe leaked; quantity injected unknown. No. 60: the dose was possibly too small; this is the only error not fully explained by experimental accidents. This reduces the percentage of failures to 1·6.

Bullocks.—No. 3: the only lesion was a *fibrous* tubercle in the lung. No. 7: there was a rise of 1° F. The animal was not well; this is not a negative but a doubtful reaction. No. 19: there was a minute calcareous tubercle in the mesenteric glands, tubercle bacilli was found, but the process was apparently becoming quiescent. It is doubtful whether any of these cases is a complete failure; two cases may be accepted as such. This reduces the percentage of failures to 2·5.

Cows.—Nos. 5, 6, 7, 19, and 33: in all these cows there was a rise of temperature ranging between 1° and 2° F. They should have been considered doubtful. No. 16 was eight years old. Nos. 27, 33, 58, and 60 were nine years old. Nos. 34, 59, and 61 were ten years old. Nos. 7 and 62 were eleven years old. Nos. 6, 17, 20, 42, and 65 were twelve years old. Nos. 5 and 19 were thirteen years old. All the failures, therefore, occurred in cows over eight years of age.

We may safely exclude from this table all the results which are vitiated by experimental faults clearly admitted. On the other hand, we must retain those results which though of doubtful value may be accepted as necessary defects which would occur under ordinary circumstances. After excluding from the animals one the age of which was not stated and five which cannot be considered as having been properly tested (in two cases old inactive tuberculin was injected; in another the syringe leaked and the quantity was not known; in two others it was doubtful whether there was active tuberculosis or not) I have constructed tables bringing out facts which had not been previously paid sufficient attention to and which corroborate my previous experience in Manchester (Table III.).

Even if the five cases which I have excluded on good grounds from the groups of animals under eight years of age were retained in that group, the total number of errors would have been nine out of 209 tests, an error equal to 4·3 per cent. I should state also that all the positive tests were found correct, whilst of the nine cases in which a negative reaction was misleading, seven were affected with extremely localised tuberculosis arrested or nearly arrested, and only two were suffering from pulmonary tuberculosis and might have been a source of infection. In this group, therefore, the amount of serious and material error amounted to two out of 209, or less than 1 per cent. With regard to the second group of aged cows, they were all in an advanced state of tuberculosis, and their state would have been easily recognised by clinical examination.

I will show later that under a proper system of inspection tuberculosis should never reach such a degree as to render tuberculin useless as a method of diagnosis. It may be interesting to note that similar experiments conducted in 1891 in the Duchy of Baden by Lydtin, according to the same plan as that adopted in Aberdeen, yielded the following results. Of 110 head of cattle there was negative tuberculin reaction in 70, in none of which were tuberculous lesions found *post-mortem*; there was positive tuberculin reaction in 39, and tuberculous lesions were found *post-mortem* in 37 of them;

TABLE III.—*Influence of Age and Disease on the Results of the Tuberculin Test.*

	Number of animals giving a definite reaction.	Tuberculin reaction.		Tuberculosis found post-mortem.	Total number of tuberculous animals.	Error on - reaction.	Error on + reaction.
		-	+				
<i>Heifers.</i>					Per cent.		
Under two years of age }	57	51	6	7	12.25	1	0
<i>Bullocks.</i>							
2 years of age .	98	78	20	22	22.44	2	0
<i>Bulls.</i>							
3 years of age .	3	2	1	1	33.33	0	0
<i>Cows.</i>							
4 years of age .	5	4	1	1	20.0	0	0
5 " " .	9	5	4	4	44.44	0	0
6 " " .	19	9	10	10	52.6	0	0
7 " " .	10	4	6	6	60.0	0	0
8 " " .	8	7	1	2	25.0	1	0
Totals for all animals up to 8 years of age }	209	160	49	53	25.4	4 = 1.91%	0
<i>Cows.</i>							
9 years of age .	6	5	1	5	83.0	4	0
10 " " .	6	5	1	4	66.4	3	0
11 " " .	4	3	1	3	75.0	2	0
12 " " .	6	6	0	5	83.0	5	0
13 " " .	3	3	0	2	66.6	2	0
Totals for cows above 8 years of age }	25	22	3	19	76.0	16 = 76%	0

and there was doubtful reaction in one animal. In this experiment the negative reactions were free from error. The error in connection with the positive reactions were two in number, but as no precaution appears to have been taken to allow the animal to rest before the injection of tuberculin, and as, perhaps, not sufficient attention was paid to deep-seated lymphatic glands, the results obtained were remarkably accurate.

The results of these three sets of experiments, which bear upon a total of 408 animals, leave, I think, no room for doubt as to the efficiency of tuberculin as a diagnostic agent when it is properly used. The amount of error attributable to failure of the test is very small;

indeed, it is trifling when compared to the errors which are inherent to most methods of diagnosis, many of which are, nevertheless, used as a basis for administrative procedure. To the direct evidence which I have already given I may add some indirect evidence which I have collected in the course of my investigations on cow's milk as a source of infection. In the course of six years nearly 2000 samples of milk have been examined in my laboratory; of these samples over 500 were from single cows. I am greatly indebted to the Manchester Sanitary Committee, and especially to Dr Niven, for the facilities which have been given to me for collecting the information which I wished to obtain; to Mr Holborn, the chief veterinary inspector; and to Mr Lloyd, the veterinary inspector attached to the Sanitary Department, for supplying me with all the material I required. I have also to acknowledge similar help received from Mr Tattersall, the medical officer of health of Salford, and his veterinary inspector, Mr Fordham. The following table records some of the data which I have thus obtained. The smallness of the numbers is undoubtedly disappointing considering the amount of trouble taken, but the absence of any discordant result renders these figures, small as they are, very significant. Most of the 505 cows, the milk of which was examined separately, had suspicious udders. A reliable record of the effects of the tuberculin test was obtained in fifty cases only. The following table (Table IV.) shows the effects of inoculation of guinea-pigs with the milk of these fifty cows.

TABLE IV.—*Information Collected in Connection with the Examination of Milk from Single Cows with Diseased Udders since 1896.*

Year.	Total number of cows examined.	Total number in which a record of the tuberculin test was obtainable.	Number of cows giving a positive reaction.	Number of cows giving a positive reaction and the milk of which was tuberculous.	Number of cows giving a negative reaction.	Number of cows giving a negative reaction and the milk of which was tuberculous.	Doubtful reaction.	Milk tuberculous.
1896-97	30	15	12	5	3	0	—	—
1898	4	0	—	—	—	—	—	—
1899	173	14	12	1	2	0	—	—
1900	208	19	12	2	5	0	2	0
1901	90	2	0	0	0	0	—	—
Total	505	50	36	8	10	0	—	—

This evidence shows that none of the cows giving a negative reaction with tuberculin yielded tuberculous milk. On the other hand, all those cows which yielded tuberculous milk gave a positive result.

PART II.—ADMINISTRATION AND FINANCE.

A. Methods which have been recommended for the Prevention of Tuberculosis in Cattle.—When one reviews the various methods which have been recommended, and sometimes carried into execution, for the purpose of checking the progress of tuberculosis, one is struck with the fact that most of these measures had for their chief object the protection of the consumer of tuberculous products, more especially meat and milk. The measures provide the means of controlling to a certain extent cattle which have become tuberculous, but they do not make, in my opinion, any adequate provision for the eradication of existing tuberculosis or for the prevention of the spread of the disease among cattle. I think that the chief causes of the failure of the legislation which has been proposed so far in various countries have been the following. 1. Nowhere has the use of a sufficiently accurate method of diagnosis been made the basis of measures taken. Tuberculin has been recommended, but option in favour of clinical methods has almost invariably been allowed. This, as I have shown, has for effect to allow a large number (probably from 80 to 90 per cent.) of tuberculous animals to remain undetected. On the other hand, if the use of tuberculin had been made compulsory as a basis of action with regard to cattle under seven or eight years of age, not more than 2 or 3 per cent. would have escaped for a time but would have ultimately been discovered on re-injection. (The proportion of aged cattle kept on farms is gradually diminishing.) 2. With the exception of the short and necessarily incomplete trials which have been made by the State of Massachusetts and the Belgian Government those methods have been partial and not general, so that what good was obtained in one farm or district has been counteracted by the absence of precautions in a neighbouring farm or district and by the shifting of tuberculous cattle from districts under control to those not supervised. 3. When slaughter of tuberculous animals has been resorted to this has generally been done without discrimination, and the necessary compensation has become so costly as to be prohibitive. These have, in my opinion, been the chief causes of failure, and I have, therefore, attempted to find if it would not be possible to discover some solution of these difficulties.

B. Experimental Stamping-out of Tuberculosis in a Small Herd.—

1. I have shown by the Ford Bank experiment how, by a careful application of the tuberculin test, disinfection of premises, and thorough disinfection, it is possible in the short space of one year to replace entirely a highly tuberculous stock by stock free from tuberculosis remaining on the original premises. This stock remained entirely free from tuberculosis during a second year. To make this clear I may sum up the results in the following way: May 1898.—Old stock: twelve milch cows and heifers, eleven tuberculous; three bullocks, two tuberculous; and six calves, all free from tuberculosis. Total, twenty-one animals, thirteen of which were tuberculous, or 61·88 per cent. February 1900.—New stock, including old stock which had not previously reacted: twenty milch cows and heifers,¹ none of which were tuberculous; one bullock, not tuberculous; and nine calves born since the beginning of the experiment,

¹ The average age of these cows was higher than in the case of the old stock.

none of which were tuberculous. Total, thirty animals, all free from tuberculosis.

2. In my experiments it has been impossible to test the effects of general measures except by the rigid exclusion from the estate of new cattle which had not passed the tuberculin test. This was done by making a negative tuberculin reaction a *sine quâ non* condition to the introduction of any fresh cattle. Twenty-four cows or heifers, apparently healthy and specially selected, were offered to Mr Ashton's steward under these conditions. They were all isolated and tested with the following results: of eleven cows, five years of age or more, six reacted, and of thirteen cows or heifers, from two to three years of age, one reacted, giving a total of twenty-four animals, seven of which reacted.

3. The financial aspect of the question, which from a scientific point of view is not the most important, is, probably, from the practical and administrative sides, the chief difficulty which has to be overcome before satisfactory preventive measures can be put in force. The Ford Bank experiment was therefore conducted in such a way as to yield as much information as possible on this point. It will, however, be obvious to those who are acquainted with farm management that it was impossible for Mr Ashton to have the experiment conducted as economically as it could have been by a practical farmer. Owing to the conditions of a farm kept in pleasure-grounds the Ashton experiments must be considered as indicating the maximum cost of preventive measures. This, however, does not in the least diminish their importance. In May 1898 the approximate value of old stock, which consisted chiefly of twenty-one well-bred Yorkshire Shorthorns (not pedigree cattle), was £262.¹ Thirteen of these gave a positive reaction at the first testing with tuberculin and were slaughtered at once—viz., eight cows, three heifers, and two bullocks, which were all found *post-mortem* to be tuberculous. Except in one case all the viscera had to be condemned; in addition to this, one whole carcase was condemned on account of generalised tuberculosis, and the half of another was also found to be unfit for food. The sum realised for the carcasses was £159, 4s. 8d., and the value of the remaining stock was £48, 10s. (total, £207, 14s. 8d.); the actual loss was therefore £54, 5s. 4d., to which must be added the veterinary charges, which amounted to £10, making in all £64, 5s. 4d. There was also a serious loss of dairy produce, which was due to the immediate slaughter, and which would not have occurred to the same extent had arrangements been made to slaughter the diseased cows only at such time when they were in proper condition and it had been possible to replace them rapidly. (The milk of diseased cows should, of course, always be sterilised before use.) The next step taken was to replace the diseased stock. The shippens having been thoroughly cleansed and disinfected new stock was bought subject to the tuberculin test—that is to say, the cows were kept at Ford Bank for about a fortnight, during which they were tested, and only those not reacting were kept. A fee of £3 was paid to the seller as a general consideration for the trouble caused by this arrangement. But I think he found

¹ The cattle were in these experiments valued by an independent expert, the value of whose opinion is generally recognised in the district.

further compensation in the prices which he charged for the cattle sold under these special conditions.

The experiment carried out by the Cheshire County Council may be compared with advantage with the Ford Bank experiment. This important experiment was conducted on very much the same lines as the Ford Bank experiment, but about one year later. The cattle tested at the county council farms at Worleston and Holmes Chapel numbered over seventy. The results are very similar to those obtained at Ford Bank.

A general consideration of the results obtained at Ford Bank, Worleston, and Holmes Chapel leaves no doubt as to the value of tuberculin as a diagnostic agent—that is to say, as far as the positive reactions are concerned. This is a matter of great importance from an administrative point of view, for tuberculin could hardly be employed for administrative purposes if it were found that many non-tuberculous cows gave a reaction similar to that given by tuberculous cows. With regard to the value of the negative reactions, I have shown in the first part of this communication that by proper attention to certain conditions they are also quite reliable. It is also evident that the immediate loss caused by indiscriminate slaughter of all tuberculous animals is considerable. Valuable stock being excluded, there was a loss of £52, 11s. 6d. on the nine tuberculous animals from Worleston—that is, a little more than £5, 16s. per head.¹ At Ford Bank the loss on the thirteen animals slaughtered was £64, 5s. 9d.—*i.e.*, a little under £5 (veterinary charges included). On the twenty-two animals from both places the average loss was nearly £5, 7s. per head.

C. Influence of Age on the Losses caused by Tuberculosis.—In order to obtain information on this point I divided all cattle into five groups: (1) five years of age and over; (2) three years and up to five years exclusive; (3) two years and up to three years; (4) one year and up to two years; and (5) under one year. Then I found for each group the number of tuberculous animals, their estimated value under ordinary circumstances as dairy stock, and their value as meat after they had been slaughtered on account of tuberculosis and thoroughly inspected at a public abattoir by competent veterinary inspectors.² I cannot give here the details of these calculations, which I have summarised in the following table (Table V.).

¹ A later estimate, based on the losses caused by the slaughter of nineteen tuberculous animals, brought the loss up to £7, 7s. per head. This would bring the average loss on the two experiments up to £6, 8s. per head. (In this later estimate, however, are included some valuable animals, the estimated value of which in life was out of proportion to the value of their carcasses; the loss on such animals should always be considered separately.)

² Information regarding the age of cows which cannot be found in the Cheshire County Council Report I have obtained through the courtesy of Mr R. P. Ward, the organising secretary to the Technical Instruction Committee.

[TABLE.

TABLE V.—*Showing Losses caused by Slaughter of Tuberculous Cattle of Various Ages.**Ford Bank.*

	Percentage of cows and heifers tuberculous.	Estimated value of 100 animals be- fore testing.	Loss on 100 such animals after slaughter of tuberculous ones.	Percentage loss on capitul.
	<i>Per cent.</i>			<i>Per cent.</i>
5 years and above	100	£1900	£735	38'00
3 years and up to 5 years . .	75	2015	622	30'86
2 " " 3 "	100	1400	<i>Nil.</i>	<i>Nil.</i>
1 year " 2 "	<i>Nil.</i>	800	<i>Nil.</i>	<i>Nil.</i>
Under 1 year	<i>Nil.</i>	320	<i>Nil.</i>	<i>Nil.</i>

Worleston and Holmes Chapel.

5 years and above	31'25	£1515	£189	12'47
3 years and up to 5 years . .	20'69	1800	240	13'33
2 " " 3 "	<i>Nil.</i>	1300	<i>Nil.</i>	<i>Nil.</i>
1 year " 2 "	<i>Nil.</i>	800	<i>Nil.</i>	<i>Nil.</i>

It is evident from these figures that if herds are to be kept free from tuberculosis by a method which will not cause unnecessary pecuniary loss, this must be done by removing all animals which have become tuberculous before they have reached their third year. Before that date the slaughter of tuberculous animals does not involve any serious loss to the farmer; at a later period the loss becomes very serious, more especially in connection with cows between the third and fifth year, when they have reached their greatest value as milkers. With regard to bullocks the problem is simpler, but it is equally evident that all tuberculous animals should be isolated before they are two or three years of age, and then slaughtered as soon as they can be profitably sold to the butcher. If all the tuberculous stock over three years were eliminated from our herds it would be quite easy for the farmer, assisted by competent veterinary inspectors, to keep his stock ever after practically free from tuberculosis without any serious expenditure to himself.

D. Practical Experiment to Test the Relative Meat Value of Tuberculous and Non-Tuberculous Cattle under Two Years of Age—In order to test whether the foregoing conclusions were correct, Mr. Gair Aitchison, generously consented to bear the cost of a new experiment. This I will call the second Ford Bank experiment. Ten barren heifers, not over two years of age were to be brought by the steward at a fair or from a cattle dealer. They were to be selected in the usual way on the basis of their external appearance and estimated value and not subject to the tuberculin test. After a few days rest they were to be tested, and the animals reacting were to be entirely separated from those non-reacting. This young stock was then to be fattened and when ready for the butcher their value as live stock was to be estimated by an expert. The animals were then to be tested again and slaughtered at the Market for carcasses and the carcasses were to be

thoroughly inspected by Mr Holburn, the chief veterinary inspector, who kindly undertook to report upon them. The carcasses were then to be offered for sale at the meat market. Mr Jones¹ was to keep a complete account of the cost of keep, special feeding, and testing, as well as the amount realised after slaughter. By this experiment I hoped to obtain definite answers to the following questions: 1. Old tuberculous cattle being removed, what would be the cost of keeping the purified herds free from tuberculosis by the slaughter of young tuberculous animals during the first few years following the first application of the stamping-out measures? 2. What loss, if any, would be incurred by the selling to the butcher of young tuberculous carcasses? 3. Would the tuberculin reaction prove a practical basis for the carrying out of the method which I had suggested? The second Ford Bank experiment was carried out according to the general plan indicated above. Circumstances, however, prevented our waiting for a time favourable to purchase on suitable terms. It was also found impossible to select the best time for fattening and selling. The ten heifers were tested a second time on 15th November, with the result that the six which reacted in July reacted again, and the four which did not react continued to give a negative reaction. They were then sent to be slaughtered at the Manchester abattoir on 22nd November. Their carcasses were there examined by Mr Holburn in the presence of Mr Locke, who had tested the cows, and Mr Jones. Mr Jones reported to me as follows: "In each case Mr Holburn was able to say which of the animals had reacted, *without any reference to the testing notes*; it was therefore perfectly clear that tuberculosis was present in all those animals that had reacted. In no case was it necessary to condemn any part of the carcass." The original cost of the ten heifers, *plus* the cost of keeping them, for four months and fattening them, had been £120, 11s. 6d. The total sum realised after slaughter was only £90, 12s. 2d. There was therefore a loss of about £3 per head. This loss was not so great as that borne in connection with cattle of all ages, which was over £5 per head; but in my opinion there should have been no loss at all—in fact, I expected a profit. There are, however, certain features of the experiment which account for the failure. 1. The cattle had been bought irrespectively of the conditions of the market, and, as I understand, too late in the season. 2. The steward who bought the cattle had not much experience of the value of live stock; this was the opinion of two experienced men who warned me before the conclusion of the experiment of the fallacy which this might introduce. It was also unfortunate that two heifers in calf should have been bought, as such animals, whatever their value might be in life, were not (before calving) suitable beasts for the meat market. As a speculation the experiment was not a success; but considering the mode of purchase and selling it could not be expected to be a sound transaction. The total sum paid for the ten animals in July was £96. Their total estimated value as living stock in November was £108, 10s. Their total cost, *plus* the cost of keeping and fattening, was in November £120, 11s. 6d. If from this total expenditure the sum of £5 spent for the carrying out the test is subtracted, there remains still a total expenditure of £115, 11s. 6d. It is therefore obvious that even if

¹ Mr Jones had charge of the cattle and dairy at Ford Bank.

tuberculosis had been out of the question there would have been a loss in the transaction due to some error in the purchase. It is also possible that too much had been spent on special feeding—£1. 1s. per head. The experiment has not, therefore, yielded the same results as it would have yielded in the hands of an experienced and skilful farmer. But although it was not successful with regard to management, it has nevertheless given very important results with regard to the relative value of the flesh of tuberculous and non-tuberculous young cattle. The following table (Table VI.) of results gives the value obtained for eight of the ten carcasses (two of them were sold together, and it was impossible to ascertain afterwards their separate value):—

TABLE VI.—*Showing the Prices obtained for Eight Carcasses.*

<i>A. Tuberculous heifers not in calf.</i>	<i>B. Tuberculous heifers in calf.</i>	<i>C. Non-tuberculous heifers.</i>
No. 45. £9, 16s. 6d. No. 46. £8, 12s. 0d. No. 51. £7, 16s. 0d.	No. 47. £7, 15s. 0d. No. 49. £7, 12s. 3d.	No. 44. £9, 1s. 6d. No. 52. £7, 9s. 0d. No. 53. £6, 7s. 0d.
Totals—£26, 4s. 6d.	£15, 7s. 3d.	£22, 17s. 6d.
Averages per head— £8, 14s. 10d.	£7, 13s. 7½d.	£7, 12s. 6d.

The same animals were also valued before being slaughtered, and it was found that during life they stood in the same relation as their carcasses did. The same prices had been paid originally for the three tuberculous heifers in Group A and the three healthy heifers in Group C. There seems, therefore, no evidence showing that a moderate amount of tuberculosis depreciates the meat value of young carcasses. As a matter of fact, in this case better value was obtained for tuberculous carcasses than for those which were free from the disease—a fact to which I will have to allude again. This matter being one of great importance, I thought it desirable to obtain an independent opinion from Mr Holburn, who had inspected the carcasses. I therefore asked him to give me without any reserve his views on the results we had obtained. With his permission I reproduce the answer:—

MARKETS DEPARTMENT, CITY ABATTOIRS,
WATER STREET, MANCHESTER.
23rd January 1900.

DEAR PROFESSOR DELÉPINE.—In answer to your inquiry as to the market value of Mr Ashton's experimental cattle and as to whether there was any difference in the conditions of those animals that reacted to the tuberculin test from those that did not react as regards their saleability, I feel quite confident in expressing the opinion that as regards their carcase value those animals that reacted came out as well as—in fact, one or two of them a little better than—those that did not react to the test.

ALFRED HOLBURN, M.R.C.V.S.,
Chief Inspector.

This valuable statement is in complete agreement with the results which I have recorded above. It is, however, evident that it would have been preferable to use animals two and a half or three years old for the experiment. The age which I had fixed was two years, in order to avoid cows in calf, but this complication was not successfully avoided. Taking everything in consideration it seems, therefore, evident that if young tuberculous cattle be properly prepared for the meat market there could not be a more profitable or convenient way to dispose of them than by that method. It is equally certain that at no other period in their life could their flesh be utilised with less danger to public health¹—a danger which should disappear entirely if private slaughter-houses were entirely abolished and all the meat submitted to thorough inspection, as it is in the Manchester city abattoirs.

E. Disposal of the Milk of Tuberculous Cows.—Whilst I consider it advisable to deal with young stock under three years of age as rapidly as possible, I think that in the present state of things the indiscriminate slaughter of all tuberculous animals above that age would be attended with considerable and unnecessary losses. Only cows in an advanced stage of tuberculosis, showing evidences of wasting or of tuberculosis of the udder, should be slaughtered without delay. They are serious sources of infection, and the longer they are kept the less their value becomes, if they retain any at all.

With regard to the cows which are not in an advanced stage of tuberculosis, it is desirable that those in milk should be fattened only after lactation had ceased or had been caused to stop. As it is impossible to foretell how soon the udder of a tuberculous cow will become diseased, it would be wise to consider the milk of all tuberculous cows as suspicious. It should therefore be sterilised before being used. With the object of finding how this suspicious milk could be utilised I made two years ago a number of experiments. I found that it was not easy to obtain butter free from abnormal taste with cream which had been heated for a few minutes to a temperature exceeding 75° C., and that, contrary to results obtained by some other observers, complete safety could not be obtained by the heating of cream up to 85° C. for fifteen minutes. Some of the results obtained are given in Table VII.

¹ I do not wish to convey by this the impression that the consumption of meat from tuberculous cattle is attended with much danger.

[TABLE.

TABLE VII.—*General Results of Experiment on the Sterilisation of Cream.*¹

	Temperature.	Duration of exposure after the temperature indicated had been reached.	Results of inoculation of guinea-pigs with equal quantities of treated and untreated tuberculous cream.
First set of experiments	74° C. (165° F.)	30 minutes	Tuberculosis slight and delayed, well-marked after three weeks.
	76° C. (169° F.)	10 "	Tuberculosis slight and delayed, well-marked after three weeks.
	82.5° C. (180° F.)	5 "	Tuberculosis nearly normal.
	85° C. (185° F.)	15 "	Tuberculosis considerably delayed (two months).
Second set of experiments	70° C. (158° F.)	45 "	Tuberculosis delayed, but well-marked after three months.
	65° C. (149° F.)	360 "	No tuberculosis after three months.
	65° C. (149° F.)	180 "	Slight tuberculosis after one month.
	65° C. (149° F.)	60 "	No tuberculosis after one month.
Controls	—	—	Well-marked tuberculosis after three weeks.

No reliance should be placed on pasteurisation of cream at temperatures up to 85° C. for the times indicated in the above table. I regret to say that in a previous paper I had expressed the opinion that a lengthened exposure of cream to a temperature below 85° C. would probably be sufficient to destroy the tubercle bacilli. I stated, however, at the same time, that I had not completed the experiments which I was making with the view of confirming the experiments of other observers.² In my opinion, therefore, the *milk of all tuberculous cows should be boiled before it is used*. I do not venture at present to suggest any special means of utilising this milk; all I wish to do is to express a *warning against an implicit belief in the statements which have been made regarding the ease with which the milk of tuberculous cows can be rendered innocuous*.

F. Innocuousness of Tuberculin Inoculations.—It has been frequently stated that the use of tuberculin was detrimental to milch cows. Not being able to investigate this matter personally I had asked Mr J. Jones to give me his impression upon the effects which our experiments had had on the dairy cows kept at Ford Bank. The following was his answer: "The quality of milk from tested stock was all that

¹ I wish to acknowledge here the cordial assistance which I received from Dr E. J. Sidebotham and Dr F. J. H. Coutts in the carrying out of these experiments, some of which lasted for a considerable time.

² See "Veterinarian," July and August, 1899.

could be desired. I do not consider testing to be at all detrimental to dairy cows in any way if carefully done." Knowing that Mr Walter W. Law of Briarcliff Manor, New York, had for some years past determined to clear his extensive herds from tuberculosis, and had had for this purpose his cows repeatedly tested with tuberculin, I asked him to supply me with some information on the effects of the testing on healthy cows. He had the great kindness to forward my request to Professor Leonard Pearson of the University of Pennsylvania, who inspects biannually the Briarcliff Farms' dairy herd. This herd is composed of over 600 "thoroughbred or high-grade" Jersey cows. Professor Pearson replied as follows:—

UNIVERSITY OF PENNSYLVANIA,
DEPARTMENT OF VETERINARY MEDICINE, PHILADELPHIA,
31st March 1900.

DEAR SIR.—In reply to the questions submitted by Professor Delépine regarding the effect of tuberculin on healthy cows I would say: 1. No effect on the flesh or the weight of the animals has been observed. The members of the Briarcliff herds, tested twice each year, are in high condition. 2. The quantity of milk usually diminishes slightly for the milking of the morning following the administration of the tuberculin. This diminution is not constant and is not greater than often occurs at other times from a change of feed, a change of weather, or other natural cause. The loss does not exceed 5 per cent. of the total product of the herd. I have the impression that this is due to the slight disturbance to which the cows are subjected during the test. The herd as a whole is remarkably productive, the yield per cow per day often exceeds 20 pounds in the winter. 3. No change in the richness of the milk has been observed. It is very rich. It is not possible at Briarcliff to compare tested and untested cows in respect to the above points, because there are no untested cows on the farm. It is clear, however, that the Briarcliff standard is extremely high in regard to all of them. I may add that the large herd (500 milch cows) of Mr Francisco in New Jersey is tested with tuberculin, and Mr Francisco states that his herd is more productive and the cows in better condition than before it was his practice to test. Mr Gordon of the Walker-Gordon Laboratory Company holds the same opinion as a result of his experience with herds aggregating more than 500 cows.

Yours very truly,

LEONARD PEARSON.

To WALTER W. LAW, ESQ., Briarcliff Manor, New York.

G. Disposal of Valuable Stock.—I would urge that pedigree cattle in which tuberculosis has not produced serious lesions should be carefully isolated and watched, that these animals should be used for breeding purposes, and slaughtered as soon as possible after they have sufficiently fulfilled the purpose for which they are kept. In the event of the disease affecting their general health they should be slaughtered without further delay.

H. Market Value of Living Tuberculous Animals.—Before concluding I must refer to an aspect of the question which the experiments previously recorded bring out. Early tuberculosis, far from affecting detrimentally the appearance of young and middle-aged stock, is often associated with all the external appearances of flourishing health. Many tuberculous cows are exceptionally good milk producers.

They have often a very good market value during life. It is not therefore easy to persuade the farmer that tuberculosis may be a serious source of loss to him. He does not consider it a matter of importance to discover whether a cow is tuberculous or not so long as the animal is a good milk producer or does not lose flesh. It is not denied that a herd affected with tuberculosis is liable to greater depreciation than a herd free from tuberculosis, but it is urged that in the present state of things the loss of capital involved by the removal of tuberculous cattle would be so great that the ultimate benefit would not be sufficient or obtained soon enough to prevent ruinous losses. It is therefore stated that farmers cannot be reasonably expected to adopt measures which would be contrary to their own interests. If the matter could be considered in the light of a commercial transaction affecting only one generation of speculators having no responsibility towards their country or humanity, the above argument might be considered sound. It is, however, the duty of the State to see that the present generation does not sacrifice for its own benefit the patrimony of future generations, thereby doing great harm to the country. It is also the duty of the State to see that any condition of things which tends to perpetuate the existence of a disease which is so detrimental to man as well as to the lower animals is checked as speedily as possible. Even if the financial argument against the stamping out of tuberculosis were as sound as it appears to be it does not prove that farmers are justified in remaining passive. If through ignorance they have allowed the evil to grow beyond control they cannot consider this a valid excuse for not doing their duty as soon as they know what their duty is. The fact, however, that the carrying out of this duty towards the State would mean complete ruin in the case of many small farmers, whose welfare is also of importance to the whole country, clearly shows that a part of the losses caused by the stamping out of tuberculosis should be borne by the State.

CONCLUSIONS.

1. Tuberculin is an almost infallible test of the presence of tuberculosis in animals under seven years of age, when this fluid is used with proper care. In older cattle clinical methods are more reliable than the tuberculin test as used at present. (The use of larger doses than the usual ones may, however, be resorted to with advantage in these cases.)
2. It is possible by the use of tuberculin and thorough disinfection of cattle-sheds to stamp out tuberculosis from a herd in the course of one year.
3. Cases of infection which may accidentally occur after this preliminary removal of tuberculous cattle can be easily and economically detected by the periodical use of tuberculin.
4. Tuberculous animals between two and three years of age can be disposed of on the meat market without serious loss.
5. The slaughter of cows in calf or of milch cows not in an advanced stage of tuberculosis involves a serious loss to the farmer, and so long as tuberculosis is as prevalent as it is at the present time it is doubtful whether the measures necessary for the extirpation of tuberculosis can be left to private efforts without State help.
6. To remove the chief source of infection immediate slaughter should be resorted to in the case of all animals in a state of advanced tuberculosis, and of all cows with tuber-

culosis of the udder. 7. Milch cows and cows in calf in which tuberculosis is not very advanced should be isolated and slaughtered only after they have been suitably prepared for the butcher. The milk of tuberculous cows should invariably be sterilised by boiling. 8. Valuable breeding animals free from advanced tuberculosis or tuberculosis of the generative organs should be isolated; they can be kept for a time for breeding purposes, provided the calves are isolated and fed on sterilised milk. 9. Measures which do not provide for the removal of all tuberculous animals from a district, for the disinfection of all cattle-sheds in that district, for the periodical testing of all the animals within the district, and which do not give the means of preventing the importation of tuberculous animals cannot be expected to yield satisfactory results.

MEASURES RECOMMENDED.

On the basis of these conclusions I feel justified in recommending once more the general measures which I have advocated on several occasions during the last few years. These may be summed up as follows:—1. Division of the whole country into a number of well-defined administrative areas each provided with an efficient staff of inspectors. 2. Marking and registration of all cattle for purposes of inspection and notification. (This would, of course, mean the inclusion of tuberculosis among the recognised contagious diseases.) Systematic periodical inspections of herds and cowsheds within each administrative area. Testing with tuberculin of all cattle. 3. Isolation of all tuberculous cattle. Disinfection of sheds which have been occupied by tuberculous cattle. 4. Immediate slaughter of all cattle in an advanced state of tuberculosis (including all cases of udder tuberculosis). 5. Fattening of all animals not in an advanced state of tuberculosis for the meat market. All cattle should be slaughtered in public abattoirs so as to insure thorough meat inspection. 6. Compensation during one year or two for losses incurred by owners of cattle, except in cases regarding which there had been gross or culpable neglect. After this transitional period of compensation the presence of a case of advanced tuberculosis in a cowshed to render the owner liable to a penalty. 7. Testing of all cattle brought into the area so as to prevent the introduction of fresh sources of tuberculosis. No cattle above six years of age to be imported. 8. All the milk from tuberculous cattle to be boiled before use. Control of all imported foreign dairy produce so as to enforce the same standard of purity as in the case of home produce. 9. To avoid a sudden national depression in the dairy industry, which would be favourable to foreign produce, I further suggest that the enforcement of these measures should not be simultaneously carried out all through the country, but that they should be at first enforced in a certain number of administrative areas, to which year after year other districts should be added, until the whole country was under administrative control. It is probable that the work could be accomplished in some five or six years.

N.B.—This paper does not deal with evidence showing that bovine tuberculosis is communicable to man; this has been done by the author in a series of papers published since 1892, vide *Medical Chronicle* (1895), *THE LANCET* (1897), etc.

IS TUBERCULOSIS OF MAN TRANSMISSIBLE TO CATTLE?¹

By Professor THOMASSEN, Veterinary College, Utrecht, Holland.

I HAVE scarcely any doubt that my communication announced on the identity of human and bovine tuberculosis will acquire much interest owing to the sensational address which Professor Koch delivered at the General Meeting on Tuesday last. The great authority on tuberculosis denies the identity in question, and his assertion is mainly based on the results obtained by the inoculation of cattle with pure cultures or sputum from consumptive men.

In the experiments carried out along with Professor Schütz he has not succeeded in setting up tuberculous lesions. At the most, there was observed at the point of inoculation some suppuration, the pus containing tubercle bacilli.

Against the conclusion drawn by Professor Koch, namely, that there is little or no danger of infection of human beings through the milk and flesh of tuberculous animals, protests were entered by Professors Nocard, Bang, and Woodhead.

Nocard mentioned that in this Congress itself the results of certain experiments in contradiction with those of Koch would be communicated and supported by proof. Hence the experiments which I now wish to bring before you. But, in the first place, I ought to give a short historical account of the question concerning the receptivity of cattle for human tuberculosis. Although Laennec had already identified human tuberculosis with the disease of the same name in cattle, for a long time afterwards, and even after the magnificent discovery of Villemin in 1865, the lesions of bovine tuberculosis were considered to be of a different character from those met with in man. For a number of years the pathologists, with Virchow at their head, defended the opinion that "grapes" of cattle is a disease entirely distinct from human tuberculosis. It was not until 1882 that their opposition was entirely overcome by the discovery of Koch's bacillus, and the demonstration of its constant presence in the specific lesions in all species.

However, during the period between 1865 and 1882 numerous attempts were made to prove the identity of the two diseases, and these were successful. The necessary proof consisted in taking the virus from the one species and causing it to develop in the other. It therefore goes without saying that there cannot be any attempt to infect human beings from animals, but if one succeeds in one of the directions the reciprocal ought to be admitted as very probable.

The first attempts were made in 1868 by Chauveau, at the Veterinary College at Lyons. The infection of animals of the bovine species with human tuberculous virus was effected in three different ways, namely, by introducing the tuberculous material into the alimentary canal, by intravenous injection, and by injection into the subcutaneous connective tissue.

Three subjects which had swallowed infective material taken from human lesions all became tuberculous. The lesions had the same

¹ Communicated to the British Congress on Tuberculosis, July 1901.

characters as those in animals which had ingested materials taken from bovine lesions.

In one case the human virus was injected intravenously. After twenty-nine days the injection had determined the formation of interesting lesions entirely localised in the chest.

The hypodermic injection of a tuberculous emulsion from a human source provoked a local tuberculosis with the lesions confined to the neighbouring lymphatic glands, and without the least manifestation of generalisation.

The author arrived at the conclusion that tuberculous human virus had exactly the same effect on bovine animals as tuberculous virus taken from animals of the same species.

Klebs inoculated a four-weeks old calf intraperitoneally with tuberculous matter from a man. When the animal was killed after three months a large number of small, pedunculated, non-calcified tumours, with the histological characters of the tubercles of natural bovine tuberculosis, were found in the omentum and on the stomach. There were also some grey miliary tubercles in the mesenteric lymphatic glands, and in the liver and spleen. Klebs does not mention any lesion at the point of injection.

Kitt obtained in a calf an acute tuberculosis and death in forty-six days by inoculation with 20 grammes of juice taken from the scrofulous glands of a man.

Bollinger appears to have observed the development of typical "grapes" after the inoculation of sputum of a consumptive human being into the peritoneum of a calf. The lungs and other thoracic organs did not present any lesions.

Crookshank inoculated a calf into the peritoneum with sputum diluted with a weak solution of common salt. After forty-two days the animal succumbed in consequence of pyæmia. Together with abscesses containing Koch's bacilli, he observed a miliary tuberculosis of the lungs and liver.

These few experiments furnish evidence that human tuberculosis may be communicated experimentally to cattle, and prove at the same time that the two diseases are identical.

The results of experimental infection with pure cultures of bacilli from human sources were notably different, and this has given rise to some conclusions which are very pernicious from a practical point of view.

The first experiments of this nature were made in 1898 by Theobald Smith of Washington. He first called attention to the morphological difference between the human and the bovine bacilli, and he then pointed out a still greater difference with respect to their pathogenic properties.

The bacilli of bovine tuberculosis were more virulent for the majority of experimental animals. Death followed more rapidly, the lesions were more extensive, and the bacilli present in them were more numerous.

Smith inoculated eleven cattle, namely, five with bovine bacilli, and six with human bacilli. Of the former, two succumbed in consequence of a miliary tuberculosis; and of the three others which were killed after two months, one was affected with typical bovine tuberculosis, and the two others had lesions which were regarded as of a

tuberculous nature. The animals suffered from fever, and rapidly emaciated during the first weeks after inoculation.

The results were different in the case of cows inoculated with cultures from human sputum. When slaughtered after two months none of these animals showed anything beyond local and insignificant lesions.

Frothingham carried out parallel experiments on four calves with a culture of bacilli obtained from the liver of a child a year old.

Calf I., aged three months, was inoculated intraperitoneally. When tested with tuberculin after five weeks the animal showed a reaction of 1.9° C. Six weeks after infection it was slaughtered.

Calf II., aged three weeks, was inoculated in the same way. When tested with tuberculin after five weeks it had a thermic reaction of 1.6° C.

Calf III., aged three weeks, was inoculated into the trachea, and when tested with tuberculin after five weeks it had a reaction of 2.9° C. It was killed six weeks after infection.

Calf IV., aged two months, was also inoculated into the trachea. After five weeks, when tested with tuberculin, it had a reaction of 1.4° C.

Three of these calves had a local tuberculosis in the neighbourhood of the point of inoculation—that is to say, in the case of those inoculated into the abdomen, on the omentum and peritoneum, and in the other (intratracheal injection) in the cervical lymphatic glands and in the muscles of the neck on the inoculated side. The tuberculosis was not generalised in any of the cases.

In three calves infected with sputum from a human being, Frothingham found after five months insignificant local lesions in two, and no lesions at all in the third.

The conclusion which the author drew from these experiments was that the calf appears to have very little susceptibility for human bacilli. Whether this diminished receptivity was a consequence of a slighter virulence of the bacilli or a greater resistance of the animal remained to be decided by more numerous experiments.

Richard Gaiser recorded an experiment in 1899. He inoculated a calf into the eye and into the subcutaneous tissue with human bacilli, which were found to be very virulent to the guinea-pig and rabbit.

The first day the eye showed symptoms of very severe irritation, but this had entirely disappeared after fifteen days. After eight days the cornea had become transparent, and one could distinguish the iris, on which there had formed a small yellowish focus. The latter gradually disappeared. A small swelling formed in the flank, but disappeared without leaving any trace. About three months after inoculation the calf was slaughtered, and no tuberculous lesions were discovered at the *post-mortem* examination.

The rabbits and guinea-pigs inoculated died in consequence of a generalised tuberculosis.

Another calf, infected in the same way with bovine bacilli, of which the virulence had been reinforced by passage through a rabbit, developed a generalised tuberculosis.

EXPERIMENTS BY THE AUTHOR.

The surprising results published in 1898 by Theobald Smith induced me to carry out similar experiments in order, if possible, to

help to clear up the question, which appeared at first to attract only the attention of bacteriologists, although it may be of the greatest practical importance from the point of view of hygiene, as we have just seen in this Congress.

For the first experiment, which was made in March 1899, two calves were used, one aged eight and the other four weeks.

They were first tested with tuberculin. With a dose of 10 centigrammes there was no reaction. One could therefore consider the animals as free from tuberculosis. The milk with which they were fed was furnished by a cow that did not react to tuberculin.

I.—The calf, aged eight weeks, was inoculated into the abdomen with a glycerinised potato culture of tubercle bacilli obtained from the exudate from a case of tuberculous meningitis in a child. The culture in question was obtained from the laboratory of Professor Spronck. Its virulence had been ascertained by experiments on the rabbit and guinea-pig.

During the first weeks after inoculation the animal remained in apparent good health.

On 14th May it was tested a second time with tuberculin. On the previous three days the average temperature had been 38.6° C.

After the injection of 0.15 gramme of tuberculin the following thermic reaction was observed:—

May 15	.	.	8 A.M.	.	.	39.7
" "	.	.	10 "	.	.	39.9
" "	.	.	12 NOON	.	.	40.2
" "	.	.	2 P.M.	.	.	40.5
" "	.	.	4 "	.	.	40.6
" "	.	.	6 "	.	.	40.0
" 16	.	.	10 A.M.	.	.	38.8

The maximum rise of temperature was 2°, and this occurred at the seventeenth hour after injection. It therefore appeared to be probable that the calf had contracted at least a local tuberculosis, especially as during the last few days the hair had somewhat lost its lustre, and the appetite was not very good. The calf was slaughtered during the first days of the month of June, and, in spite of a most minute *post-mortem* examination, no trace of a tuberculous lesion was discovered.

II.—The four-weeks-old calf was inoculated at the beginning of April. After having undergone the before-mentioned preliminary test, it was inoculated into the anterior chamber of the eye with a culture on glycerinised potato, obtained from a case of tuberculous arthritis of the human subject. The virulence of the bacilli was ascertained by experiments on small animals. From the day afterwards symptoms of an intense keratitis showed themselves, and the cornea became so opaque that it was impossible to observe the alterations that were taking place on the iris.

Meanwhile the animal was attacked with enteritis (white scour), which required serious treatment. During convalescence a certain degree of paresis was manifested, the animal almost constantly retaining a recumbent position. Notwithstanding this, the appetite was retained.

On 14th May the animal was again tested with tuberculin. The average temperature during the three preceding days had been 39° C.

After the injection of 0·15 gramme of tuberculin at about 10 P.M., the following temperatures were observed next day :—

May 15,	8 A.M.	.	.	.	.	39·5
" "	10 "	.	.	.	.	39·6
" "	12 NOON.	.	.	.	.	39·1
" "	2 P.M.	.	.	.	.	39·1
" "	4 "	.	.	.	.	39·0
" "	6 "	.	.	.	.	38·9

The maximum rise of 0·5° seemed to exclude the existence of tuberculous lesions.

On 27th May, about six weeks after infection, the calf was slaughtered, and found to be the subject of a pretty generalised tuberculosis.

The eyeball was markedly atrophied. After hardening, sections of it showed tuberculous lesions, especially on the iris, these lesions containing masses of Koch's bacilli. The subparotideal, cervical, mediastinal, and bronchial lymphatic glands of the same side were tumefied and hypertrophied. On section they showed miliary tubercles. The bacilli were recovered from the bronchial lymphatic glands, and sections of the various glands showed tuberculous lesions containing the bacilli. The lungs were affected on both sides, especially at their apices.

On their surfaces there were observable some greyish-yellow elevations, which, on section, were found to be about the size of a nut, and to contain caseous material. In cover-glass preparations made from this material there were observable bacilli very similar morphologically to Koch's bacilli, but they were decolourised by the method of Ziehl-Gabbet.

In addition, the lungs contained numerous miliary tubercles, and some grey fibrous tubercles of a larger size. Microscopic examination of these showed that they had the histology of tuberculous lesions and contained the specific bacilli.

None of the tubercles showed any trace of calcification.

The *post-mortem* examination thus showed that the case was one of pretty generalised tuberculosis. The path followed by the bacilli from the eye to the lungs was evident.

III.—In the month of December 1899 a two year old heifer, in excellent condition, was used for another experiment.

In the month of November, when tested with 0·25 gramme tuberculin, the animal's temperature had risen only 0·5° C.

On the 1st December a rich culture on potato was injected into the animal's trachea. The culture had been obtained from the tuberculous kidney of a man. No symptom of disturbance followed, and on 13th February 1900, when the animal was again tested with 0·30 gramme tuberculin, the maximum rise of temperature did not exceed 1° C.

On 16th February the whole of a culture was injected into the thorax, and another into the abdomen. Of the latter a small quantity escaped into the muscles of the left flank.

On the following day the temperature had risen to 40·3°. The animal was dull, and there was very little appetite. After two days these symptoms had disappeared. A firm tumour, as large as the fist and easily displaced, gradually formed in the flank at the point of

inoculation. After three weeks this was exposed, and after incision of a fibrous envelope as thick as one's finger a yellowish purulent material escaped. The latter was rich in the specific bacilli, and these were obtained in culture. The cultures were characterised by a pale colour. The wound, which was not treated by antiseptics, gradually cicatrised, and soon only a slight fibrous enlargement remained.

In the month of July 1900 the tuberculin test was again repeated without producing a notable reaction. The heifer was slaughtered in the month of April 1901. It was in excellent condition, and not the slightest tuberculous lesion was found in it.

IV.—On the 1st December 1899 a fourth heifer, aged about two years, and which had also previously been tested with tuberculin, was inoculated into the anterior chamber of the right eye with bacilli cultivated from the kidney of a man. The following day the cornea was opaque, but not to such a degree as to prevent one from observing, two days later, the presence of an exudate in the anterior chamber. The animal appeared to suffer a good deal from this, and the appetite was diminished. Nevertheless, the temperature remained normal.

On the 19th December its condition had become aggravated, the thermometer showing a temperature of 40.6° C., falling to 40.4° C. the same evening. The fever persisted for three days. During this time the eyeball had increased to double the normal size, and, in consequence, the eyelids were no longer able to completely cover the cornea, which therefore became dry and rough.

On the 13th February 1900 the tuberculin test was repeated with 0.30 gramme. Before injection the average temperature had been 39.0° C.

The temperatures shown below were recorded on the following day :—

Feb. 14,	7 A.M.	.	.	.	.	39.5
" "	8 "	.	.	.	.	40.3
" "	9 "	.	.	.	.	40.1
" "	11 "	.	.	.	.	40.0
" "	12.30 P.M.	.	.	.	.	39.7
" "	2 P.M.	.	.	.	.	40.0
" "	3 "	.	.	.	.	39.7
" "	6 "	.	.	.	.	39.7

The maximum rise had thus been 1.3° . It was also observed that the animal was dull, its head being depressed, and the appetite diminished. There was increased swelling of the eye, and of the peri-orbital region. The animal coughed a good deal. All these symptoms led us to believe that the heifer was tuberculous.

About the 15th May following it was slaughtered. There were some tuberculous lesions in the eye, and also some tubercles in one of the bronchial glands, but, unfortunately, these were not submitted either to a histological or bacteriological examination.

Conclusions.—It follows from the experiments that it is difficult, but not impossible, to set up in the bovine species a generalised tubercu-

losis by means of pure cultures of bacilli obtained from the human subject. That the two diseases are identical still remains a firmly established truth, from which it follows that we must always take into account the danger of infection from cow to man, which, in my opinion, is more likely to happen than the reciprocal infection.

Contrary to Koch's opinion, the generally admitted higher virulence of bovine bacilli justifies us in exercising a strict supervision of the milk and flesh of tuberculous animals.

The success of experimental infection depends in large measure on the degree of resistance offered by the animal, as Nocard has suggested, and as is demonstrated by the second experiment.

It is desirable that numerous experiments may be carried out as soon as possible, and we do not doubt that the result will prove that the theory founded on the experiments of Shütz and Koch is erroneous, and the outcome of a premature conclusion.

BACTERIOLOGICAL EXAMINATION OF THE LESIONS IN A SERIES OF HORSES THAT HAD REACTED TO MALLEIN.

By J. M'FADYEAN, Royal Veterinary College, London.

DURING the latter half of 1890 and the beginning of the current year I had the opportunity to make a *post-mortem* examination of a number of horses that were supposed to have been affected with glanders. These animals formed part of the series which are referred to in Mr Spencer's report to the London County Council.¹

The animals belonged to studs in which clinical cases of glanders had been detected, and they became objects of suspicion in consequence of a reaction to mallein. Under an arrangement with the London County Council the reacting horses were removed to separate stables, and, while allowed to work as usual, they were re-tested with mallein at intervals of about a month. As soon as any of the animals developed clinical symptoms of glanders they were killed, and finally, to terminate the experiment, the horses that continued to react although apparently healthy, and some of those that had ceased to react, were killed and submitted to *post-mortem* examination, in order to determine whether they were actively glandered or not.

For the records of the testing of the animals I am indebted to the County Council's Veterinary Inspectors, Mr Hunting and Mr Humphrey.

Case I.

History.—This mare formed part of a stud that was tested with mallein in the month of May 1900, and although it did not react the

¹ See last number of this Journal, p. 169.

owner preferred to stable it with the reactors because it had been in intimate contact with glandered animals. The mare was tested a second time on the 16th June, and a third time on the 7th July. On the first of these occasions the highest temperature recorded was 101.5° , and on the second 102.5° . The latter temperature was accompanied by only a small swelling at the point of injection.

Autopsy.—The mare was killed on the 13th July, and portions of the lungs were brought to me for examination on the evening of the 14th (Saturday). These contained a number of nodules with the naked eye characters of glanders lesions.¹

Experiments.—Material taken from the centre of one of these nodules with precautions against accidental infection was used on the same evening for the inoculation of sterilised potato. On the forenoon of the 16th (Monday) two guinea-pigs were inoculated subcutaneously from two other tubercles, and a third was inoculated by scarification.

One of the guinea-pigs inoculated subcutaneously died on the 18th July, and the other on the 20th, both from malignant œdema starting from the point of inoculation.

On the 25th July the guinea-pig inoculated by scarification (on the right posterior quarter of the abdominal wall), was found to have an ulcer at the seat of inoculation, and the adjacent precrural gland was swollen. It was killed, and material scraped from the centre of the gland (which showed some white necrotic areas) was smeared on the surface of sterilised potato. On incubation this yielded a thin white growth of micrococci, and some colonies of glanders bacilli.

The potato inoculated on the 14th July from one of the pulmonary nodules yielded a continuous mixed growth, including what appeared to be glanders bacilli. On the 23rd July two guinea-pigs were inoculated subcutaneously from this impure growth. The guinea-pigs were killed on the 14th August, and typical glanders lesions were found in both. Glanders bacilli in pure culture were obtained from the lesions in one of them (no attempt to do so was made in the other case).

Remarks.—This case illustrates the advantage of inoculating by scarification rather than by subcutaneous injection when the parts to be examined are partially putrid. As is well known, the malignant œdema bacillus, which is a common organism in putrid organs, is certainly and rapidly fatal to the guinea-pig by subcutaneous injection, but infection with the organism generally fails when the material containing it is merely rubbed into the lightly scarified skin. In this case inoculation by scarification obviated infection with the accidentally present malignant œdema bacillus, but it did not prevent a mixed infection of the resulting ulcer and of the neighbouring lymphatic gland.

From the clinical point of view little is to be learned from the case. Twice within the two months preceding its death the animal had failed to react to mallein, but an indecisive rise of temperature followed the test made six days before death. It appears to be not improbable that the mare was healthy when first tested, and subsequently contracted the disease from some of the reacting horses with which it was stabled.

¹ In subsequent cases, for the sake of brevity, lesions which had the appearance of glanders nodules are referred to as "glanders nodules."

Case II.

History.—This gelding had been thrice tested with mallein, viz. :—

<i>Date.</i>	<i>Max. temp.</i>
May 19, 1900	104·8
June 6, „	104·4
July 22, „	104·4

On each occasion there was a large local reaction. Between the dates of the second and third tests the horse developed clinical signs of glanders, and it was killed on the 24th July.

Autopsy.—The lesions observed were as follows: Lymphangitis of the near hind leg, with three farcy buds on the hock. Several subcutaneous unopened farcy buds on the near side of the neck. In the lungs, several pea-sized dark-red glanders nodules, a walnut-sized lesion with central curdy contents, and a few pin-head firm grey nodules. One small white nodule in a bronchial gland.

Experiments.—A potato tube inoculated from one of the pulmonary nodules yielded a pure growth of glanders bacilli.

Three guinea-pigs inoculated subcutaneously on the 24th July from pulmonary nodules were killed on the 14th August. Two of them showed typical glanders lesions, including an ulcer at the seat of inoculation and suppuration of the adjacent precrural gland. A pure growth of glanders bacilli was obtained from one of them (no cultures attempted from the other). The third guinea-pig was quite healthy.

Remarks.—An interesting point in this case is the escape of one of the three guinea-pigs inoculated from the pulmonary nodules. In explanation of this result it may be suggested either that the lesion used for inoculation was not a glanders nodule, or that it had become inactive and no longer contained living glanders bacilli. A third possibility ought perhaps to be admitted, viz., that such bacilli were not entirely absent, but the number introduced under the skin was not sufficient to determine infection.

The case is illustrative of the great value of mallein for diagnosis, as the horse was in excellent condition and presented no indication of glanders until after the second test.

Case III.

History.—This gelding was tested with mallein on four occasions :—

<i>Date.</i>	<i>Max. temp.</i>
May 5, 1900	104
„ 20, „	102
June 6, „	103·2
July 22, „	103·8

On each occasion there was a large local reaction (6 to 10 inches in diameter).

The horse was killed on the 24th July on account of recently developed clinical symptoms.

Autopsy.—The off fore limb was swollen from the knee to the elbow, and pus was discharging from an orifice behind the last-named joint. On dissection the subcutaneous tissue of this region was found to be considerably thickened, and in it were some small

abscesses (size of hazel nut) with thick yellow pus. A few glanders nodules were present in the lungs. In a cover-glass preparation from the subcutaneous pus, stained with methylene blue, no bacteria of any kind could be distinguished with certainty.

Experiments.—A potato tube inoculated with pus from one of the subcutaneous abscesses yielded a pure culture of glanders bacilli. A guinea-pig inoculated subcutaneously with the same material on the same date (24th July) was killed on the 18th September, and the *post-mortem* examination showed neither local nor visceral lesions. Another guinea-pig inoculated subcutaneously on the same date from one of the pulmonary nodules was also found to be healthy when it was killed on the 18th September.

Remarks.—The discrepant results of the cultural and inoculation experiments are difficult to explain. As in some of the subsequent cases the negative results of the attempts to infect guinea-pigs suggests caution in drawing conclusions unless a considerable number of animals have been inoculated.

Case IV.

History.—This gelding was placed in company with reactors for the same reason as Case I. It had been thrice tested with mallein, as shown below :—

<i>Date.</i>	<i>Max. temp.</i>
May 30, 1900	101·8
June 16, „	103
July 7, „	103·5

On the second and third of these occasions there was a considerable local reaction (maximum diameter 6 inches).

The animal was killed on the 28th July on account of recently developed symptoms of farcy.

Autopsy.—On the course of the left jugular vein there were three subcutaneous firm nodules, each about the size of a horse bean. Pus was present towards the centre of the nodules, but the skin over them was intact. The off fore leg was swollen from the fetlock to the knee, and on the outer aspect of the fetlock some ulcerating sores were present. On the off cheek, a few inches above the angle of the mouth, there was a subcutaneous nodule as large as a horse bean, and discharging at its summit. In the region of the hip on either side a firm nodule as large as a horse bean was present in the subcutaneous tissue. These also were suppurating at the centre. The lungs contained about a dozen glanders nodules, some being quite typical, with a red hæmorrhagic zone.

Experiments.—Pure cultures of glanders bacilli were obtained in potato tubes inoculated from pulmonary nodules and from the farcy lesions over the left jugular vein.

A guinea-pig inoculated subcutaneously on the 28th July from one of the pulmonary nodules was found to be healthy when killed on the 14th August. Another inoculated from a different nodule developed typical glanders lesions. A similar positive result followed in two other guinea-pigs inoculated respectively from a nodule over the jugular vein and a nodule in the region of the hip. Pure cultures of glanders bacilli were obtained from these three guinea-pigs.

Remarks.—Here, again, the negative result in one of the guinea-pigs inoculated from a pulmonary nodule deserves to be noted.

Case V.

History.—The subject in this case was a mare which had been four times tested with mallein, viz. :—

<i>Date.</i>					<i>Max. temp.</i>
May 26, 1900	.	.	.	.	104·6
June 16, "	.	.	.	.	104
July 7, "	.	.	.	.	103
" 28, "	.	.	.	.	103

On the first and third tests there was a large local reaction (5 or 6 inches); on the second and fourth it was one-half smaller.

Early in August the mare developed clinical symptoms, and it was slaughtered on the 9th of that month.

Autopsy.—This revealed numerous glanders nodules in the lungs, ulceration of the nasal septum, and farcy lesions on both hind legs.

Experiments.—Pure cultures of glanders bacilli were obtained on potato from one of the farcy lesions. A guinea-pig inoculated on the 10th August from four pulmonary nodules mashed together was killed on the 7th September, and found to have an ulcer at the point of inoculation, and suppuration of the adjacent precrural gland. Another guinea-pig inoculated on the 10th August with a trace of farcy pus was also killed on the 7th September. It had similar lesions, with, in addition, nodules in the spleen. Glanders bacilli in pure culture were obtained from one of the guinea-pigs (no cultures made from the other).

Case VI.

History.—This was a mare which had been four times tested with mallein :—

<i>Date.</i>					<i>Max. temp.</i>
May 26, 1900	.	.	.	.	105
June 16, "	.	.	.	.	103·5
July 7, "	.	.	.	.	104·5
Aug. 4, "	.	.	.	.	104

On each of these occasions there was a considerable local reaction (4 to 6 inches).

The mare was destroyed on the 16th August on account of recently developed farcy lesions.

Autopsy.—Two farcy ulcers were present on the off fore leg, and glanders nodules were present in the lungs and bronchial glands.

Experiments.—A pure culture of glanders bacilli on potato was obtained from one of the pulmonary nodules. A guinea-pig inoculated subcutaneously on the 16th August from another pulmonary nodule was killed on the 6th September. The *post-mortem* examination showed an ulcerating sore at the point of inoculation, suppuration of one of the testicles, and numerous minute white nodules in the spleen. A potato tube inoculated from the testicular pus yielded a pure growth of glanders bacilli.

Case VII.

History.—This gelding had been twice tested with mallein:—

<i>Date.</i>	<i>Max. temp.</i>
July 19, 1900	104
" 29, "	104

The diameter of the local swelling on both these occasions was about 3 inches.

The animal was killed on the 17th August on account of recently developed farcy lesions.

Autopsy.—The lesions comprised three farcy sores on the off hind leg, swelling of the deep inguinal glands on the same side, and a few glanders nodules in the lungs.

Experiments.—Two potato tubes inoculated from pulmonary nodules yielded very mixed growths, and no attempt was made to separate the different species.

A guinea-pig inoculated subcutaneously on the 18th August from a pulmonary nodule died on the 10th September. There was an ulcer at the point of inoculation, and the adjacent precrural and inguinal glands were suppurating. The testicles were normal, and there were no visceral lesions. A potato tube inoculated with pus from the inguinal gland yielded a pure culture of glanders bacilli.

Remark.—The apparently healthy condition of the testicles of the guinea-pig three weeks after infection is an exceptional feature of the case. Possibly the guinea-pig's death was not altogether attributable to the glanderous infection, and had it survived a little longer the testicles would doubtless have been involved.

Case VIII.

History.—This gelding was tested with mallein as follows:—

<i>Date.</i>	<i>Max. temp.</i>
July 7, 1900	104·8
" 28, "	104·5
Aug. 18, "	102·8

On the occasion of the first and second tests there was a large local reaction (6 inches), but it was only half that size on the 18th August.

The animal developed clinical symptoms of glanders, and was slaughtered on the 31st August.

Autopsy.—This revealed ulceration of the nasal septum, tumefaction of the submaxillary glands, and numerous glanders nodules in the lungs and bronchial glands.

Experiments.—Potato tubes inoculated from lung nodules yielded colonies of glanders bacilli mixed with some impurities.

Two guinea-pigs were inoculated subcutaneously from a pulmonary nodule on the 31st August. The material was prepared by mashing the nodule with about 5 cc. of sterile water, and 1 cc. of the liquid was used for each guinea-pig. Both were killed on the 12th October. One of them (a male) was quite healthy. The other (a female) had an ulcerating sore at the point of inoculation, suppuration of the adjacent precrural gland, and two pin-head white nodules in the

spleen. Two potato tubes inoculated with pus from the precrucial gland yielded pure growths of glanders bacilli.

Remark.—That one of the guinea-pigs escaped infection is somewhat remarkable. The fact emphasises the importance of using several experimental animals in all doubtful cases.

Case IX.

History.—This gelding had been thrice tested with mallein:—

<i>Date.</i>	<i>Max. temp.</i>
July 7, 1900	103·8
" 28, "	104·5
Aug. 18, "	103

The local swelling had a diameter of 6 inches on the first and second tests, and of 3½ inches on the third.

The animal was killed on the 31st August on account of clinical manifestations of glanders and farcy.

Autopsy.—The lesions comprised farcy buds, nasal ulceration, tumefaction of the submaxillary glands, and a considerable number of glanders nodules in the lungs. Some nodules were also present in the bronchial glands.

Experiments.—Glanders bacilli were cultivated in a state of purity from pulmonary nodules. On the 31st August two guinea-pigs (a male and a female) were inoculated, as in the previous case, from a pulmonary nodule. They were killed on the 12th October, and both were found to be quite healthy.

Case X.

History.—This subject was a mare that had been five times tested with mallein:—

<i>Date.</i>	<i>Max. temp.</i>
July 30, 1900	104·5
Aug. 25, "	103·4
Sept. 15, "	103
Oct. 6, "	102·8
Nov. 3, "	102

On the 30th July and 6th October there was a large local swelling (5 or 6 inches), but on the other occasions the local reaction was slight.

The mare was killed on the 10th November on account of symptoms of acute pleurisy.

Autopsy.—This revealed a moderate number of glanders nodules in the lungs and a single pin-head nodule in one of the tracheal group of lymphatic glands. The parietal and visceral pleura was thickly coated with fibrinous lymph.

Experiments.—On the 10th November six nodules cut out from the lungs were triturated in a mortar with 10 cc. of sterile bouillon, and two guinea-pigs were subcutaneously inoculated with the liquid, each receiving 1½ cc. Both animals died on the 12th November, with an extensive putrid inflammatory lesion around the point of inoculation. Fortunately the lungs of the mare had not been destroyed, and on the same date four other pulmonary nodules were prepared as above

and used to inoculate three guinea-pigs, each animal receiving 1 cc. of the turbid bouillon. At the same time the entire pin-head lesion in the tracheal gland was introduced into a subcutaneous pocket on the abdominal wall of a fourth guinea-pig.

Two of the animals inoculated from the pulmonary nodules were killed on the 27th November, and in both there were present an ulcerating sore at the point of inoculation, suppuration of the adjacent precrural gland, and some small white nodules in the spleen. One of the animals was a male, but its testicles were normal. A potato tube was inoculated from the suppurating precrural gland of each guinea-pig, and a pure growth of glanders bacilli was obtained.

The third guinea-pig inoculated from pulmonary nodules died with similar lesions on the 15th December.

The guinea-pig inoculated with the nodule in the tracheal gland was not killed till the 11th May 1901, and it was then found to be healthy.

Remarks.—Unfortunately in this case no attempt was made to obtain cultures from the pleural lymph, or to determine whether the pleurisy was of a glanderous nature. An interesting feature in the case was the steady decline of the intensity of the reaction under repeated tests with mallein although the animal remained actively glandered.

Case XI.

History.—This gelding had been tested with mallein as follows:—

<i>Date.</i>	<i>Max. temp.</i>
May 19, 1900	105
June 6, "	103·8
July 22, "	102
Aug. 18, "	101·6
Sept. 9, "	102
Oct. 20, "	103
Nov. 11, "	100

The local swelling on the occasion of the first test measured 5 inches in diameter, but on the later dates it was small or almost entirely absent.

The animal was slaughtered on the 13th November 1900.

Autopsy.—Only five glanders nodules could be detected in the right lung and two in the left, the largest being of the volume of a vetch pea. The right bronchial gland contained a white nodule about the size of a pin's head, and a similar lesion was present in a tracheal gland. There were some coarse thread-like fibrous growths on the surface of the liver, and a few white, completely calcified nodules under its capsule. Some soft thread-like growths were present on the surface of the spleen. The submaxillary, pharyngeal, and abdominal lymphatic glands were normal, as were also the nasal mucous membrane and the kidneys. The skin was free from farcy lesions.

Experiments.—On the 14th November three of the pulmonary nodules were torn out with sterile instruments, and triturated separately with a few cc. of bouillon. Three guinea-pigs were subcutaneously inoculated—one from each tubercle, and the quantity of liquid injected was 1 cc. All these nodules had firm fibrous capsules and central dry, but non-calcified contents.

A fourth guinea-pig was inoculated in the same manner from the nodule in the tracheal gland.

All the guinea-pigs were killed on the 10th December, and all were found to be healthy.

Case XII.

History.—This gelding had been tested with mallein as follows:—

<i>Date.</i>	<i>Max. temp.</i>
April 28, 1900	103·4
May 20, "	101·6
June 6, "	103·4
July 22, "	102
Aug. 18, "	101
Sept. 9, "	101·4
Nov. 11, "	101

The local swelling was large on the occasion of the first two tests (5 or 6 inches), but on the later dates it was small or absent.

The horse was killed on the 13th November.

Autopsy.—On the right side of the chest-wall, corresponding in position to the 9th, 10th, and 11th ribs in their lower third, there was a flat swelling about 6 inches in diameter. At its summit the skin was perforated by a small opening, around which the hair was matted with discharge. On dissection this swelling was found to be mainly composed of inflammatory fibrous tissue, with some small collections of pus in it. A careful search revealed only two nodules in the right lung and none in the left. One of the tracheal glands contained a pin-head white nodule. There were some fibrous thread-like growths on the anterior surface of the liver, and one completely calcified white nodule under its capsule.¹

Experiments.—On the 14th November the two lung nodules were triturated with bouillon (as in the preceding cases), and used for the inoculation of two guinea-pigs. In tearing out the tubercles it was observed that one of them was rather ill-defined and lacked contents as distinct from capsule, but the other, which had the volume of a barley grain, had distinct dry, firm, cheesy contents.

The lesion in the tracheal lymphatic gland was similarly used to inoculate a guinea-pig. It was found to be calcified.

A fourth guinea-pig was inoculated with pus from the lesion on the chest-wall. A potato and an agar tube were also inoculated with this material.

The potato tube inoculated with pus developed no visible growth, but the agar one yielded a crop of small circular colonies of bacteria of the fowl cholera type.

One of the guinea-pigs inoculated from the lung nodules was found dead on the 7th December. The seat of inoculation and all the organs were free from macroscopic lesions, and no bacteria were discoverable in the blood. The cause of death was not ascertained.

The remaining three guinea-pigs were killed on the 10th December, and found to be normal.

¹ To save space in most of the cases the parts examined for glanders lesions with negative results are not mentioned. In every case the examination extended to the nose and throat, submaxillary and pharyngeal lymphatic glands, and to the abdominal as well as the thoracic organs.

Case XIII.

History.—A mare which had been nine times tested with mallein :—

<i>Date.</i>	<i>Max. temp.</i>
May 26, 1900	103·9
June 16, "	101
July 7, "	101·2
" 28, "	102·5
Aug. 25, "	100·4
Sept. 15, "	101·2
Oct. 6, "	103·6
Nov. 3, "	101·4
Dec. 3, "	102·2

On the occasion of the first test the local swelling was 5 inches in diameter, and on the second it measured 4 inches by 3. On all the subsequent dates it was small or absent.

The mare was killed on the 10th December on account of an injury to the spine.

Autopsy.—Subcutareously, 2 inches below the stifle joint, there was a nodule about as large as a garden bean, with a little pus towards its centre.

About a dozen nodules were found in the lungs, varying in size from a vetch pea to a pin's head.

The tracheal group of lymphatic glands contained two nodules, one as large as a barley grain and the other about the size of a pin's head. On the surface of the spleen and liver there were some villous fibrous growths, and a few pin-head white, completely calcified nodules were present under the capsule of the liver.

Experiments.—On the 10th December four of the largest lung nodules were torn out and triturated with 6 cc. of bouillon. From the resulting liquid three guinea-pigs were inoculated, 1 cc. each, and at the same time two potato tubes were inoculated with a loopful of the liquid.

The potato tubes yielded pure growths of glanders bacilli. Two of the guinea-pigs died on the 18th December. In one there were scattered pin-head white nodules under the peritoneum, and a sub-lumbar gland on the left side was suppurating. The great omentum was crammed with nodules.¹ The other guinea-pig had a large suppurating lesion at the point of inoculation, swelling of the adjacent precrural gland, and one pin-head nodule in the spleen. Pure cultures of glanders bacilli on potato were obtained from both guinea-pigs. The third guinea-pig inoculated from the lung tubercles died on the 28th December with glanders lesions involving the seat of inoculation, precrural glands, spleen, and testicles.

Inoculation and cultural experiments, which it appears to be unnecessary to describe in detail, were also made with the tracheal and hepatic lesions, and with the pus from the nodule near the stifle. In all these cases the results indicated the absence of glanders bacilli. The pus yielded yellow colonies of a micrococcus (*staphylococcus aureus* or *botryomyces*).

Remarks.—This case is important as showing that when an animal

¹ Probably the liquid used for inoculation in this case was introduced directly into the peritoneal cavity, and not under the skin as was intended.

is frequently tested with mallein at short intervals it may cease to react although remaining glandered.

Case XIV.

History.—This mare had been tested eight times with mallein:—

<i>Date.</i>	<i>Max. temp.</i>
July 7, 1900	103°6
" 28, "	106
Aug. 18, "	103°6
Sept. 8, "	103
Oct. 6, "	103
Nov. 11, "	104
Dec. 3, "	102°5
" 30, "	101°2

On the first two occasions the local swelling was 6 inches in diameter, but with the later tests it was small (2-3 inches).

The mare was killed on the 16th January 1901.

Autopsy.—The lungs contained some dozens of nodules, mostly about the size of a barley grain. Several white nodules were present in the tracheal glands. All these had the macroscopic characters of glanders nodules. Some thread-like fibrous growths were present on the anterior surface of the liver.

Experiments.—Six of the lung nodules, taken at random, were torn out and mashed with 10 cc. of bouillon. Two guinea-pigs received 1 cc. each of the turbid liquid thus obtained, and a loopful of the same was used to inoculate three potato tubes.

One of the nodules in a tracheal gland was similarly mashed with 2 cc. of bouillon, of which 1 cc. was injected into a guinea-pig.

The potato tubes developed some colonies of a micrococcus, but none of glanders bacilli.

The three guinea-pigs were killed on the 8th February, and found to be normal.

Case XV.

History.—This mare had been tested ten times with mallein.

<i>Date.</i>	<i>Max. temp.</i>
May 26, 1900	105
June 16, "	103°2
July 7, "	103°5
Aug. 4, "	102
" 25, "	103°2
Sept. 15, "	103
Oct. 6, "	104°6
Nov. 3, "	102
Dec. 3, "	102°5
" 29, "	102

On the occasion of the first test the local reaction was very small, but on the second it was considerable (5 inches by 2½). On all the later occasions it was small.

The mare was killed on the 19th January 1901.

Autopsy.—The left submaxillary gland was enlarged and contained several white tough nodules, about the size of barley grains. The lungs contained about a dozen nodules, mostly smaller than a barley

grain. Behind the heart the right lung, for the size of two fists, was in a condition of fibroid pneumonia. The left bronchial gland contained several white nodules, varying in size from a pin's head to a lentil seed, and with tough non-calcified contents.

Experiments.—One of the pulmonary nodules was torn out in its entirety, and inserted in a subcutaneous pocket on the abdominal wall of a guinea-pig. Three others, taken at random, were mashed with 3 cc. of bouillon, and after two potato tubes had been inoculated from the liquid 1 cc. of the remainder was injected under the skin of a guinea-pig.

Three nodules from the bronchial glands were mashed with 2 cc. of bouillon, and 1 cc. of the liquid was injected under the skin of a guinea-pig. Two potato tubes were also inoculated from the liquid.

A nodule from the submaxillary gland was used in the same way to inoculate a guinea-pig and two potato tubes.

A fifth guinea-pig was inoculated with a scraping from the cirrhotic patch of lung suspended in bouillon.

None of the potato tubes yielded any glanders growth, though several developed colonies of micrococci.

The guinea-pig inoculated from the bronchial gland was found dead on the 31st January, and the one from the submaxillary gland on the 7th February. In neither case was the cause of death ascertained, but the seat of inoculation, the testicles, and the viscera were macroscopically normal. The other three guinea-pigs were killed on the 8th February and found to be normal.

Case XVI.

History.—A mare that had been tested ten times with mallein:—

<i>Date.</i>	<i>Max. temp.</i>
May 26, 1900	103
June 16, "	102
July 7, "	102·2
" 28, "	103·8
Aug. 25, "	101·8
Sept. 15, "	102·4
Oct. 6, "	104
Nov. 3, "	102·6
Dec. 3, "	102·5
" 29, "	103

On the occasion of the first test the local swelling was very small, and on the later occasions it was variable, but never exceeded 4 inches in diameter.

The mare was killed on the 19th January 1901.

Autopsy.—The lungs contained over a dozen nodules, varying in size from a large pin's head to a barley grain. There was also present, in the left lung, a nodule larger than a common pea. The contents of this were greyish-brown and rather dry. The left bronchial gland contained one tough white nodule as large as a barley grain.

Experiments.—The contents of the pea-sized nodule in the left lung were rubbed up with a little bouillon and injected under the skin of a guinea-pig. A potato tube was inoculated with the same material.

Five of the remaining pulmonary nodules were mashed with 5 cc. of

bouillon, and from the liquid two potato tubes and two guinea-pigs were inoculated (1 cc. each).

The nodule in the bronchial gland was used in the same way to inoculate a potato tube and two guinea-pigs.

Some of the potato tubes developed colonies of micrococci, but none of glanders bacilli.

All the guinea-pigs were killed on the 8th February, and found to be normal.

Case XVII.

History.—A gelding which had been tested ten times with mallein :—

<i>Date.</i>	<i>Max. temp.</i>
May 29, 1900	102·4
June 16, "	101·5
July 7, "	103
" 28, "	100·5
Aug. 25, "	100·4
Sept. 15, "	101·8
Oct. 6, "	102
Nov. 3, "	100·4
Dec. 3, "	100·5
" 29, "	102·2

Except on the second occasion, when it measured 6 inches in diameter, the local swelling was small.

The animal was killed on the 21st January 1901.

Autopsy.—Only three nodules could be discovered in the lungs, the largest being about the size of a vetch pea. One of the bronchial glands contained a nodule as large as a barley grain. Some patches of thickened (fibroid) pleura were present on the costal aspect of the lungs.

Experiments.—Part of the contents of the largest pulmonary nodule was used to inoculate two potato tubes. The remainder and the two other tubercles were mashed with 3 cc. of bouillon, which was then divided equally between three guinea-pigs by subcutaneous injection.

The nodule in the bronchial gland was mashed with 2 cc. of bouillon, and 1 cc. of the liquid was injected into a guinea-pig.

The potato tubes remained sterile, and the three guinea-pigs were found to be normal when they were killed on the 8th February.

Case XVIII.

History.—A mare that had been tested ten times with mallein :—

<i>Date.</i>	<i>Max. temp.</i>
May 26, 1900	105·6
June 16, "	104
July 7, "	103·6
" 28, "	102
Aug. 25, "	102
Sept. 15, "	103
Oct. 6, "	102·8
Nov. 3, "	102·6
Dec. 3, "	102·5
" 29, "	102

The maximum local reaction was on the occasion of the first test, when it was 4 inches in diameter.

The mare was killed on the 21st January 1901.

Autopsy.—The lungs contained some hundreds of nodules varying in size from a pin's head to a barley grain. All of these had a well-defined outline, and a firm "shotty" consistence. In all those that were large enough to be divided with a scalpel it was possible to distinguish a capsule and contents, the former being grey and translucent, and the latter opaque. In the right lung, behind the heart, there was an area of chronic interstitial pneumonia, about as large as one's fist. Some thread-like fibrous growths were present on the anterior surface of the liver and on the base of the spleen.

Experiments.—Three potato tubes were inoculated with material from the centre of as many lung nodules. Ten other nodules, taken at random, were mashed with 5 cc. of bouillon, and after two potato tubes had been inoculated from the liquid two guinea-pigs each received 1 cc. of the same under the skin. A third guinea-pig was similarly inoculated with a scraping from the piece of fibroid lung suspended in bouillon.

Two of the potato tubes inoculated from separate lung nodules developed colonies of glanders bacilli, one being a pure growth, and the other having some impurities. One of the tubes inoculated from the ten mashed nodules remained sterile, and the other developed a single colony of a sarcina.

All the guinea-pigs were killed on the 8th February. Two of them inoculated from the mashed nodules were glandered, having a suppurating lesion at the point of inoculation and pus in the adjacent precrural gland (bilateral in one). From each of these guinea-pigs two potato tubes were inoculated (pus of a precrural gland), and all four tubes developed pure growths of glanders bacilli.

The third guinea-pig inoculated from the pulmonary nodules, and the one done with a scraping from the fibroid lung tissue, were both normal.

Remark.—That one of the three guinea-pigs should have escaped infection while the other two inoculated from the same material became glandered, is remarkable.

Case XIX.

History.—A gelding that had been ten times tested with mallein:—

<i>Date.</i>	<i>Max. temp.</i>
May 30, 1900	105
June 16, "	104.5
July 7, "	103.8
" 28, "	102.5
Aug. 25, "	100.5
Sept. 15, "	103
Oct. 6, "	104
Nov. 3, "	102.4
Dec. 3, "	103
" 29, "	104

The largest local reaction was on the first occasion, when it was 4 inches in diameter.

The gelding was killed on the 22nd January 1901.

Autopsy.—Only six nodules could be found in the lungs; they varied from a vetch pea to a barley grain in size. The pleura showed some patches of fibrous thickening on the costal aspect of the lungs. The left bronchial gland contained one white nodule. Some villous fibroid growths were present on the anterior surface of the liver and on the spleen, and the hepatic tissue was slightly cirrhotic.

Experiments.—The entire contents of the nodule in the bronchial gland was smeared on the surface of a potato. Part of the contents of one of the pulmonary nodules—thick and caseo-purulent in character—was divided between the two potato tubes, and the remainder was rubbed up with 2 cc. of bouillon, the half of which was then injected under the skin of a guinea-pig. The remaining five pulmonary nodules were mashed together with 4 cc. of bouillon, and of this 1 cc. was injected into each of two guinea-pigs.

The three guinea-pigs were killed on the 9th February, and found to be normal.

None of the potato tubes developed any growth of glanders bacilli. The one from the bronchial gland developed about twenty saffron-yellow colonies of a micrococcus. One of the tubes from the pulmonary nodule remained sterile, and the other developed a single white colony of a micrococcus.

Remarks.—The experiments, as will be observed, although they were as searching as possible in the circumstances, entirely failed to detect the presence of living glanders bacilli in the lesions. In view of the temperature record, however (see last test), doubts may well be entertained as to whether the animal had really recovered from glanders.

Case XX.

History.—A gelding which had been ten times tested with mallein:—

<i>Date.</i>	<i>Max. temp.</i>
May 28, 1900	104
June 16, "	104
July 7, "	103·6
" 28, "	102·5
Aug. 25, "	102·2
Sept. 15, "	101·6
Oct. 6, "	101
Nov. 3, "	101·2
Dec. 3, "	100·2
" 29, "	101·4

The only large local reaction was on the 16th June, when it measured 8 inches by 3.

The animal was killed on the 22nd January 1901.

Autopsy.—The most painstaking search revealed only two nodules in the lungs. The visceral pleura showed some white patches of fibroid thickening. There were some thready growths on the liver and spleen, and several white nodules under the capsule of the former organ.

Experiments.—One of the pulmonary nodules was pricked and found to have caseo-purulent contents, part of which was spread on

the surface of potato. The remainder and the other nodule were mashed with 4 cc. of bouillon, of which 1 cc. was injected under the skin of each of three guinea-pigs.

On dissection it was found that the hepatic nodules also had caseo-purulent matter towards their centres, and such matter from two of them was used to inoculate two potato tubes.

The three guinea-pigs were killed on the 9th February, and found to be normal. Both the tubes inoculated from the hepatic nodules remained sterile. A single yellow colony of a micrococcus was the only growth on the tubes from the pulmonary nodule.

Remark.—In this case the gradual loss of power to react to mallein and the negative result of the search for glanders bacilli were in agreement, and indicated that the animal had really recovered from glanders.

Case XXI.

History.—This was a mare that had been thrice tested with mallein:—

<i>Date.</i>	<i>Max. temp.</i>
Nov. 3, 1900	103
Dec. 3, „	102.6
„ 29, „	101.4

The local reaction was considerable (3 inches by 5) on the first occasion, but on the last it was almost absent.

The mare was killed on the 23rd January 1901.

Autopsy.—The lungs contained about a dozen nodules, varying in size from a vetch pea to a barley grain. The base of the left lung showed a cirrhotic area nearly as large as a cocoa-nut, and there were several fibroid patches on the pulmonary pleura. The anterior surface of the liver showed several “milk-spots” and some fibrous villous growths. In the substance of the liver, close beneath the capsule, there were several white, completely calcified nodules.

Experiments.—Three potato tubes were inoculated with part of the contents of two pulmonary nodules. The remainder of these two nodules and four others taken at random were mashed with 5 cc. of bouillon, of which 1 cc. was injected under the skin of each of three guinea-pigs.

A scraping from the fibroid area of the left lung was suspended in bouillon and injected under the skin of a fourth guinea-pig.

One of the potato tubes remained sterile, and the other two developed some colonies of a yellow sarcina.

The whole of the guinea-pigs were killed on the 12th February. One of those inoculated from the lung nodules (a male) was normal, while the other two were glandered, having ulcerating sores at the seat of inoculation and suppurating precrural glands. One of them also had bilateral suppurative epididymitis, and the other (a female) had some miliary lesions in the spleen. The guinea-pig inoculated from the fibroid lung had similar lesions. Glanders bacilli in pure culture were obtained from each of these three animals.

Remarks.—Here, again, the escape of one of the guinea-pigs deserves to be noted. The case is an important one, since it demonstrates that it would be highly dangerous to accept non-reaction as proof of recovery when the mallein test is repeated at short intervals.

Case XXII.

History.—This gelding had been tested ten times with mallein:—

<i>Date.</i>	<i>Max. temp.</i>
May 26, 1900	103
June 16, "	102
July 7, "	103
" 28, "	102
Aug. 25, "	100·8
Sept. 15, "	102·6
Oct. 6, "	103·4
Nov. 3, "	100·2
Dec. 3, "	103
" 29, "	102·4

On the occasion of the first test the local swelling was 5 inches in diameter, and on the last it measured 6 inches by 3. On the other occasions it was small or absent.

The animal was killed on the 23rd January 1901.

Autopsy.—The lungs contained some dozens of nodules, mostly about the size of common pins' heads. The pulmonary pleura carried a considerable growth of fibrous threads. The anterior surface of the liver and the outer surface of the spleen showed similar growths.

Experiments.—Six of the pulmonary nodules were mashed together with 5 cc. of bouillon, which was then used to inoculate four guinea-pigs (1 cc. each) and two potato tubes.

Both of the potato tubes yielded an abundant growth of micrococci, but no glanders bacilli.

One of the guinea-pigs (a female) was found dead on the 12th February. It had an ulcerating lesion at the point of inoculation, suppuration of the precrural glands on both sides, and pin-head lesions in the liver and spleen. Glanders bacilli in pure culture were obtained on potato from a precrural gland and from the liver.

The other three guinea-pigs were killed on the 12th February, and found to be normal.

Remarks.—It will be observed that this animal had entirely failed to react on two occasions (25th August and 3rd November), although the experiments proved that it was glandered at the time of death. Again it deserves to be noticed that only one out of four guinea-pigs inoculated with the same material became infected.

Case XXIII.

History.—A gelding that had been nine times tested with mallein:—

<i>Date.</i>	<i>Max. temp.</i>
May 19, 1900	106
June 6, "	104·4
July 22, "	103·8
Aug. 18, "	103·4
Sept. 9, "	105
Oct. 20, "	104·4
Nov. 18, "	103·5
Dec. 9, "	103
Jan. 20, 1901	104

On the first three occasions the local reaction was 5 or 6 inches in diameter, but on the last three it was about half that size.

The animal was killed on the 29th January 1901.

Autopsy.—The lungs contained some scores of nodules, varying in size from a filbert nut downwards. In all that were incised it was possible to distinguish a capsule and greyish-white, rather dry, cheesy contents. In the larger ones such contents formed the bulk of the nodule. A number of pin-head white nodules were present in the bronchial and tracheal glands.

Some thready fibrous growths were present on the anterior surface of the liver, and there were some partially calcified nodules under the capsule.

Experiments.—The cheesy contents were dug out of three of the lung nodules and smeared on the surface of potato—one tube for each nodule. Three other lung nodules were mashed together with 5 cc. of sterile water, and 1 cc. of the liquid was injected under the skin of each of three guinea-pigs.

Two of the potato tubes remained sterile, but the third developed four characteristic colonies of the glanders bacillus.

The three guinea-pigs were killed on the 20th February, and in each of them the seat of inoculation and the adjacent glands were healthy. In one of them there were some rather large nodules (size of a barley grain) in the spleen, but inoculation of potato tubes proved that these were not of a glanderous nature.

Remarks.—The results of the experiments were unexpected. It was thought that there would be no difficulty in proving that living glanders bacilli were present, in view of the continued reactions to mallein and the large number of characteristic lesions present in the lungs. It will be observed, however, that the experiments very nearly failed to reveal the presence of the specific bacilli, a positive result being yielded by only one tube. The experiments afford strong evidence that while some of the lesions remain active, others may have ceased to contain living bacilli although retaining their ordinary macroscopic characters. And if that be so, it is evident that it would be necessary to test a great many nodules before concluding that all of them were inert.

Case XXIV.

History.—A gelding that had been seven times tested with mallein:—

<i>Date.</i>	<i>Max. temp.</i>
April 21, 1900	105
May 20, "	103·4
June 6, "	103
July 22, "	103
Aug. 18, "	101·6
Sept. 9, "	101·4
Jan. 20, 1901	103

On the first four occasions the local reaction varied from 4 to 6 inches in diameter, but on the last two it was absent.

The horse was killed on the 29th January 1901.

Autopsy.—The left lung contained about a dozen nodules, one being nearly as large as a filbert nut, and the others smaller than

barley grains. The liver showed a "milk-spot" and some fibrous threads on its anterior surface, and about half a dozen firm white nodules beneath its capsule.

Experiments.—Part of the cheesy contents of the largest lung nodule was smeared on potato, and the remainder, together with five other nodules, was mashed with 5 cc. of sterile water, of which 1 cc. was injected into each of three guinea-pigs.

The potato tubes remained sterile, and the guinea-pigs, when killed on the 20th February, were found to be free from glanders.

Case XXV.

History.—A gelding that had been eight times tested with mallein :—

<i>Date.</i>	<i>Max. temp.</i>
July 7, 1900	102·2
" 28, "	102·5
Aug. 25, "	101
Sept. 15, "	103·2
Oct. 6, "	103·2
Nov. 3, "	101
" 3, "	103
Dec. 29, "	103·6

The largest local swelling was on the first occasion when it measured 4 inches in diameter.

The animal was killed on the 23rd January 1901.

Autopsy.—The left submaxillary lymphatic gland was thrice the size of the right; on section its tissue was pale, but devoid of nodules or other macroscopic lesions. Only five nodules could be found in the lungs; these varied in size from a common pin's head to two barley grains. On the outer surface of the spleen there was a nodule nearly as large as a hazel nut, and containing thick pus towards its centre. The anterior surface of the liver carried a patch of fibroid villous growths.

Experiments.—The five lung nodules were mashed with 4 cc. of bouillon, with which two guinea-pigs (1 cc. each) and two potato tubes were inoculated.

Two potato tubes were smeared with pus from the splenic lesion, and the remainder was suspended in bouillon and injected into a guinea-pig.

Another guinea-pig was inoculated with a scraping from the enlarged submaxillary gland, similarly suspended in bouillon.

Three of the potato tubes remained sterile, and the fourth (from lung nodules) developed a single white spot of growth.

All the guinea-pigs were found to be normal when killed on the 12th February.

Case XXVI.

History.—This gelding was seven times tested with mallein :—

<i>Date.</i>	<i>Max. temp.</i>
April 21, 1900	105·4
May 20, "	104·4
June 6, "	104
July 22, "	103·4
Aug. 18, "	102
Sept. 9, "	102·4
Jan. 20, 1901	103

On the occasion of the first test the local reaction was only 3 inches in diameter, but on the second it measured 8 inches, and on the third 5 inches by 6. Subsequently it did not exceed 3 inches, and on the last two occasions it was absent.

The animal was killed on the 30th January 1901.

Autopsy.—Only four nodules could be found in the lungs, none larger than a barley grain. Two small calcified lesions were present in one of the tracheal lymphatic glands. The only other lesions observed were some fibroid villous growths on the surface of the liver, and one pinhead completely calcified nodule under its capsule.

Experiments.—The four pulmonary nodules and the two in the tracheal gland were mashed together with 5 cc. of bouillon, and three guinea-pigs (1 cc. each) and two potato tubes were inoculated with the liquid.

One of the potato tubes remained sterile, and the other developed an abundant yellow growth of micrococci.

The guinea-pigs were found to be free from glanderous lesions when killed on the 20th February.

Remarks.—It will be observed that in this and the preceding case the animal had reacted to the last test, the temperature rising to 103° in the one case and to 103·6° in the other. Nevertheless, the experiments entirely failed to prove that the lesions contained living glanders bacilli. In view, however, of the near approach to failure in some of the other cases in which positive evidence was obtained, it would be rash to affirm that the horses had entirely recovered from the disease.

Conclusions.

The experiments were not intended to test the accuracy of mallein as it is ordinarily employed, for on that point the officers of the Local Authority in London have during the past few years had an experience which places the reliability of the test beyond any doubt. At the same time, it may be pointed out that the experiments do afford further testimony to the great value of that agent for the detection of glanders in its early stages. These horses were all free from any outward symptom of the disease when they were first put aside in consequence of having reacted, but eight of them during the next six months developed clinical symptoms, and were experimentally proved to have been glandered at the time when they were killed. In six others glanders lesions that were proved to be in an active state were discovered *post-mortem*, and in all the remaining animals the autopsy revealed lesions that had all the macroscopic characters of glanders nodules.

It may, of course, be objected that macroscopic inspection is not a certain method of determining whether a given lesion is glanderous or not, and that in some of the cases in which the cultural and inoculation experiments failed, the animal was not and never had been glandered, the lesions being of another nature. To such an objection one could only reply that the explanation suggested is possible, but highly improbable. No doubt further evidence on this point could have been obtained by subjecting the lesions to microscopic examination, but it must be remembered that the main object of the *post-mortem* examination and experiments was to determine whether living glanders bacilli were present in the animal or not.

To have used the lesions for microscopic purposes, when only a few were present might have furnished stronger proof of their glanderous nature, but it would have seriously diminished the evidence that it was the primary object of the experiments to obtain, for one cannot use the same nodule both for microscopic examination and for cultural or inoculation experiments.

It was assumed that each of the horses had been affected with glanders at the time when it first reacted to mallein, and the special object of the experiments was to ascertain whether living glanders bacilli were present in them at the time when they were killed. This, of course, had its great interest in those cases in which there had been a failure to react to some or all of the later tests. It would have been very satisfactory if the inoculation and cultural experiments in all such cases had yielded negative results, but, as will be observed, glanders bacilli were recovered from the lesions in several animals that had entirely failed to react to some of the later tests. It is therefore quite evident that when a horse reacts distinctly to the first test, non-reaction to subsequent tests repeated at monthly intervals cannot safely be accepted as evidence of complete recovery.

It ought not to be overlooked that in some of the cases the animal had had at least a temperature reaction when last tested, although the results of the cultural and inoculation experiments were negative. Probably in these cases the recovery was not complete, for it cannot be pretended that such experiments can ever be so searching as to preclude the chance of failure.

The experiments furnish no evidence at all regarding the alleged curative effect of mallein. Some of the horses apparently recovered, but fourteen of them were proved not to have recovered, and in the case of the former it is quite possible that the course of the disease would have been the same if they had never been tested.

EDITORIAL ARTICLE.

THE CAUSE OF TUBERCULOSIS, AND THE FALLACIES OF REASONING FROM ANALOGY.

ELSEWHERE in this number of the *Journal* there appears an article by Mr Goodall which, as the author explains, was primarily intended as a contribution to the proceedings of the recent British Congress on tuberculosis. Mr Goodall has some regrets that circumstances interfered with this intention, for he thinks it might have been of assistance in solving the problem set to the scientific world by Professor Koch. We have given it a place in these columns, not because we share the author's opinions, or agree with his estimate of the probable usefulness of giving them publicity, but rather because its appearance here gives us the opportunity to point out the mischief of some views

regarding the causation of tuberculosis that are, we fear, held by a good many besides Mr Goodall.

Mr Goodall admits that the opinions which he puts forward are not in harmony with what he calls the "popular scientific" opinions of the day. It is not easy to understand what is meant when two terms that are generally used in opposition are compounded to form a new adjective, but we would fain hope that some of the views which we are about to criticise are not popular, and they certainly are not scientific.

We may pass lightly over the supposed analogies which the author has culled from the natural history of the animal parasites, and also over the curious apple experiments. The author himself admits that the latter are simple and childish, and with that estimate everyone who takes the trouble to read the account of them will agree. It has been our duty to read the text several times, but it has not been possible to extract from it a glimmer of useful purpose in the experiments, or to draw from the result any conclusion that has the remotest bearing on the etiology of tuberculosis in animals or men.

Coming next to the analogies which are drawn from the sphere of bacteriology, it will be observed that much is made of the fact that the majority of the bacterial diseases of the lower animals are not transmissible, or are rarely transmitted, to human beings. Only a mind of a peculiar constitution can see in this any evidence bearing on the question of the transmissibility of tuberculosis from animals to men, and no one who wishes to escape the charge of dishonesty or culpable forgetfulness should employ the fact in argument without mentioning the numerous exceptions to the rule. It is perfectly true that neither swine fever, bovine pleuro-pneumonia, equine influenza, nor canine distemper, is transmissible to man, but it is equally true, and of great importance when the fact is used as an argument against the inter-communicability of human and bovine tuberculosis, that not one of these diseases is common to two of the domesticated species, whereas tuberculosis is naturally transmitted to horses, pigs, cats, and dogs, as well as to cattle, and is experimentally transmissible to at least a score of other animals.

The true analogies in this connection are not those cited by Mr Goodall, but such diseases as rabies, glanders, anthrax, actinomycosis, and ring-worm. Each of these resembles tuberculosis in being transmissible to several of what are called the lower species, and it is a striking fact that each of them is also transmissible to the human subject. The unbiassed searcher after truth, the earnest student of what goes on in nature's great workshop, to use Mr Goodall's own expression, ought surely to mention that fact when he is laying before his fellow men the analogical evidence bearing on the disputed identity of human and animal tuberculosis. Instead of hinting the inference that these two diseases are different because men do not

contract strangles from horses, ought he not rather to point out that every known bacterium that is pathogenic to two of the domesticated species is also pathogenic to the human subject, that bovine tuberculosis is certainly transmissible to all the ordinary domesticated quadrupeds, and that, in the absence of evidence to the contrary, it may reasonably be inferred that the bovine disease is also transmissible to man?

Passing away from this point, we have next to notice the opinions which Mr Goodall holds regarding tuberculosis as a disease of the bovine species. Here we encounter a venerable error put forward as if it were a newly discovered fact and the outcome of great clinical acumen. We are asked to believe that "tuberculosis is a cause of invasion" by the tubercle bacillus. Taken literally, this is, of course, sheer nonsense, but probably what was meant was that tuberculosis is caused not by the tubercle bacillus but by a peculiar weak state of the animal body. We are asked to believe that the only individuals successfully attacked by the tubercle bacillus are the weakly, or those predisposed by heredity, and that in-and-in breeding and breeding from immature parents are factors of great importance in the causation of the disease. In considering these statements it will be well to separate the alleged influence of heredity from the other predisposing causes enumerated.

It is scarcely possible to doubt that the degree of resistance offered by the animal body to invasion by the tubercle bacillus varies from individual to individual, and it is in the highest degree probable that animals which possess powers of resistance above or below the average will tend to transmit them to their progeny. But it does not follow from this admission that hereditary transmission of a marked predisposition to tuberculosis is a factor of great practical importance in the dissemination of the disease, or one that presses for notice when we are considering means of prevention. Moreover, it may confidently be asserted that there are not forthcoming in this country any observations which prove that inherited predisposition has been of great importance in the causation of tuberculosis among cattle. No doubt plenty of instances could be cited to prove that the progeny of tuberculous bulls and cows have themselves become tuberculous, but we know no well-authenticated case in which, with equal exposure to risks of infection, the disease has attacked the progeny of the tuberculous and spared the descendants of the non-tuberculous.

There are several reasons why it is futile to insist upon the importance of hereditary transmission of a predisposition to tuberculosis in advising cattle-owners how to get rid of the disease. In the first place, we cannot with any certainty recognise either the special predisposition or the powers of resistance that are above the average, for neither in the human nor in the bovine race is infection by the

tubercle bacillus proof of an unusual degree of susceptibility. There is always a possibility that the animal attacked may have had powers of resistance quite up to or even above the average, and that its infection where others escaped was entirely due to its having taken into its body a greater number of specific bacilli. At the present day it ought not to be necessary to point out that the belief in a phthisical type of conformation, as indicative of a special predisposition to tuberculosis, was founded on an error, and that what was taken to be the evidence of predisposition was in reality the effect of the disease. No one who has had much experience in testing herds with tuberculin can have any doubt on this point, for in numerous instances the test shows that individuals with the most faultless conformation have contracted the disease, while others that are less perfect and outwardly less robust in appearance have escaped.

What has just been said regarding inherited predisposition as a cause of tuberculosis applies for the most part to the other factors to which Mr Goodall attaches so much importance. In-and-in breeding and breeding from immature parents may tend to deterioration of the progeny in more ways than one, but it is absurd to pretend that they can be held accountable for any considerable proportion of the cases of tuberculosis occurring at the present time. There are thousands of herds badly affected with the disease in which neither in-and-in breeding nor breeding before maturity has been practised, and in which one cannot point to the operation of "exposure, hardship, over-work, or disease" as predisposing causes of tuberculosis. The one all-important factor in the immense majority of cases is the opportunity for infection—the neglect of the precautions which are necessary in dealing with a disease which has only one cause, viz., the transmission of the specific bacteria from the affected to the healthy.

We are asked to believe that an animal with a sound skin and mucous membrane is able to resist disease-inducing parasites where an inoculated one becomes a ready prey to them, and it is fair to assume that this is intended to apply to infection with the tubercle bacillus. This is another venerable error, unsupported by an atom of proof. It is perfectly certain that animals in the most robust health can be successfully infected with tuberculosis by experimental methods which closely imitate the natural, and there are no grounds whatever for believing that the animals which do not become infected in natural circumstances owe their escape to the possession of a sound skin and mucous membrane. It is probable that, other things being equal, animals and men kept in good hygienic conditions possess stronger powers of defence against the majority of the pathogenic bacteria than those whose general health is from any cause visibly deteriorated, but it is not an ascertained fact that what may be called the higher degrees of resistance to tuberculosis generally go along with the outward signs of vigorous health.

The veterinary surgeon who, when he is consulted as to the means by which tuberculosis may be eradicated from a herd, tells the owner that he must seek protection by building up the constitution of his animals, and by adopting methods of breeding that will ensure to them sound bodies and perfect mucous membranes, is offering "empty chaff well meant for grain." The man whose sense of proportion is not hopelessly defective will attach only minor importance to such considerations, and will concentrate his efforts against the one great cause of the disease—the operation of contagion. When that has been successfully grappled with predisposition becomes of no account, and as long as it is neglected the endeavour to stay the progress of the disease by any other method is as hopeless as the search for the philosopher's stone.

Review.

Annual Reports of Proceedings under the Diseases of Animals Acts, etc., for the year 1900.

THE report of the Chief Veterinary Officer to the Board of Agriculture deals with the incidence of the contagious diseases of animals during the past year, and, naturally, foot-and-mouth disease occupies the first place and comes in for the greatest share of attention. An account is given of the Board's efforts to deal with the invasion which began in January 1900, and this is followed by a useful description of the symptoms of the disease, illustrated by a series of excellent photographs of lesions encountered in recent outbreaks. The Assistant Secretary contributes a report which also deals with the operations of the Board in connection with the scheduled diseases of animals. A large part of it is devoted to swine-fever, and may be not unfairly described as an attempt to vindicate the methods which the Board has been employing against this disease with such indifferent success during the past six years. The difficulties in the way of eradication are partly inherent, largely the fault of pig-owners and dealers, but in no way attributable to any defects in the methods employed by the Board. This is doubtless a comforting view to those who are responsible for the inception and carrying out of the measures that have proved so inefficient, but it is to be feared that a good many people will remain unconvinced to it.

The reports are supplemented by the usual statistical tables, and the whole may be obtained for the modest sum of one shilling.

CLINICAL ARTICLES.

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A CASE OF BROKEN KNEES WITH OPEN CARPAL JOINT.

By JOHN PENBERTHY, F.R.C.V.S., Royal Veterinary College, London.

THE subject whose left knee is represented in the accompanying illustrations, is an aged dun carriage gelding, admitted to the College Infirmary on the 22nd May last, with the following history. On being driven through a village in Kent on the 8th April the animal stumbled and fell, its left knee coming in contact with the edge of the pavement kerb, sustaining very severe injury and an extensive wound—"as though the front of the knee had been torn off." The gelding was immediately placed in a stable close by, under the care of the local veterinary surgeon, whose name I regret I have, up to the present, been unable to ascertain, so that I cannot report the details of the earlier treatment. I am, however, informed that the wound had been kept clean by bathing with hot water and dressed with antiseptic lotions, that lameness was considerable for some days after the accident, and that there was a very small amount of discharge from the wound, but no appreciable systemic disturbance. The owner, wishing to have the patient nearer his home, had him brought to the College in an ambulance.

Examination revealed a very extensive, granulating wound, involving nearly the whole of the front of the knee, which, on being bent, showed a very deep transverse fissure, exposing to view the synovial



surfaces of the bones forming the middle (hinge-like) articulation. There was very little discharge, evidence of pain, or inflammatory action in the synovial membrane or bone. Temperature, pulse, etc., were normal.

Though one could hold out little hope of recovery to usefulness, on

learning from the owner that the animal was a great favourite, and considering the absence of local and systemic disturbance, it was decided to try the effect of surgical treatment for a few weeks. Orders were given to sling the patient, but, on account of restiveness, this was found impracticable, and to prevent lying down the head was tied short. The wound was thoroughly washed with a 2½ per cent solution of carbolic acid, and then the knee bent and a similar solution sprayed into the cavity, a pledget of cotton wool placed on the knee and a bandage applied over it. The parts were dressed in this way on alternate days till 10th June, by which time the wound was considerably smaller and without appreciable discharge. Weak solution of chinosol was now substituted for carbolic acid solution, and a dry dressing of starch, boracic acid, and oxide of zinc dusted on, cotton wool and bandage being applied as before. On the 17th and 18th a little walking exercise was given. On the 19th the patient was let loose in his box, and moved freely about until the 27th, when a fairly firm scab was accidentally removed, probably by lying down. For the five days following the animal was kept standing when in the stable, but taken daily for walking exercise. The healing process continued favourably, and on the 16th August the patient was discharged from the Infirmary, and I am informed by his owner that for the past four weeks the animal has performed his ordinary carriage work as well as before the accident.

A REMARKABLE CASE OF CONGENITAL LIPOMA IN A CALF.

By J. M'FADYEAN, Royal Veterinary College, London.

THE annexed photograph (Fig. 1) shows the appearance of the head of a nine-months'-old calf that was recently forwarded to the Royal Veterinary College as a curiosity. In the centre of the forehead, between the orbits, there was a hemispherical prominence nearly as large as a coconut, and below this, over the upper part of the nasal bones, there was a smaller swelling about the size of a pigeon's egg. On reflecting the skin, the upper swelling was found to be for the most part composed of a tumour-like mass of dry, lobulated, adipose tissue. The lower enlargement had a similar structure, with a larger admixture of fibrous tissue. On the right half of the upper mass there was a fibrous-looking portion, and on dissection this was found to be part of the wall of a cavity which corresponded to a large defect in the cranial bones, mainly involving the right frontal. The floor of the cavity presented a number of somewhat polypoid-looking, soft, grey growths, and between these the handle of a scalpel passed readily to a depth of 3 inches backwards and upwards towards the centre of the cranial cavity. There was a trace of liquid showing in this space. When the fatty tissue had been cleaned away from the forehead, the frontal bone on the left side, opposite the defect on the right side, was found to present an abnormal, thick, prominent ridge. On removal of the bones, so as to expose the brain, the left hemisphere appeared to be normal, but the before-mentioned polypoid, grey masses were

found to correspond to the roof of the right hemisphere, and were manifestly composed of cerebral tissue. The passage previously noticed was found to lead between these into the right lateral ventricle. As far as could be made out, the objects at the base of the brain and the cerebellum were normal, but the animal had been



FIG. 1.

poleaxed through the right side of the occiput, and the cerebellum and posterior part of the cerebrum on that side were partially disorganised.

Fig. 2 shows the appearance of the defect in the cranial bones after they had been freed from the soft tissues by boiling.



FIG. 2.

On subsequent inquiry it was learned from the owner that the swelling on the forehead was present at birth, and that the defect in the skull was then easily recognisable on manipulation. The slightest blow on the swelling caused the calf to fall down, but it soon got up again. Although it could move for some distance in a straight line,

it always inclined to turn to the right side. Its vision also appeared to be defective, especially for objects straight in front.

It appears to be probable that the primary mischief was the development of a lipomatous growth on the forehead during foetal life, and that this interfered with the development and ossification of the cranial vault. It may be mentioned that the mass of adipose tissue, save on its deep aspect, had all the characters of a lipoma, being sharply defined and easily separable from the adjacent parts, but it was intimately adherent to the bones by its deep face.

VENTRAL HERNIA COMPLICATED BY ACCIDENTAL ENTEROTOMY.

By T. H. RIDLER and F. HOBDAV, London.

THE patient, a female cat about eighteen months old, was admitted into our infirmary on the 27th June last suffering from a ventral hernia. The latter was as large as an average-sized orange, and situated in the left flank, immediately in front of the inguinal region. The hernia could only be partially reduced.

Under the influence of A. C. E. mixture a small incision was made through the skin and a director inserted. With a scalpel the orifice was enlarged, but, unluckily, owing to the adhesion between the sac and the skin, the former was cut through and a piece of the small intestine incised transversely for fully four-fifths of its circumference.

A number of ascarides were removed from the interior, the parts were carefully swabbed with sterilised cotton wool and chinosol lotion, and the gut was sutured with a double row of Lembert's sutures (fine silk). The adhesions of the intestine to the skin were broken down (not without some difficulty and only by the aid of the scalpel), the intestines were returned into the abdomen, and the wall of the latter was sutured with silkworm gut. The skin was sutured in the same way and treated with iodoform collodin.

The patient was kept on milk alone until the 2nd August, when a little fish was given; and, with the exception of a small abscess under the skin, it has made a perfect recovery, being sent home on the 25th July. No bad sequelæ have been observed, and the cat is in excellent condition at the present time (October).

Reports.

ROYAL VETERINARY COLLEGE.

LIST OF BURSARIES, MEDALS, CLASS PRIZES, ETC., 1900-1901.

Coleman Prizes.

<i>Silver Medal</i>	.	.	.	.	.	Mr F. W. Robards.
<i>Bronze Medal</i>	.	.	.	.	.	Mr E. C. Webb.
<i>Certificate of Merit</i>	.	.	.	.	.	Mr S. F. G. Pallin.

Centenary Prizes (£20 each).

<i>Class A</i>	.	.	.	.	.	Mr E. Brown.
<i>Class B</i>	.	.	.	.	.	Mr A. S. Leese.
<i>Class C</i>	.	.	.	.	.	Mr J. Craig.
<i>Class D</i>	.	.	.	.	.	Mr F. W. Robards.

Royal Agricultural Society's Medals.

<i>Silver Medal</i>	.	.	.	.	.	Mr J. Harrison.
<i>Bronze Medal</i>	.	.	.	.	.	Mr F. W. Robards.

Clinical Prizes.

Class A.

<i>First Prize £5</i>	.	.	.	.	Mr J. R. Hodgkins.
<i>Second Prize £3</i>	.	.	.	.	Mr D. Quinlan.
<i>Third Prize £2</i>	.	.	.	.	Mr F. V. Steward.

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Class B.

<i>First Prize £5.</i>	.	.	.	.	Mr F. Hewson.
<i>Second Prize £3</i>	.	.	.	.	Mr H. Mason.
<i>Third Prize £2</i>	.	.	.	.	Mr G. G. Brown.

Class C.

<i>First Prize £5.</i>	.	.	.	.	Mr G. Sutton.
<i>Second Prize £3</i>	.	.	.	.	Mr H. E. Gibbs.
<i>Third Prize £2</i>	.	.	.	.	Mr H. B. Hiles.

Class D.

<i>First Prize £5</i>	.	.	.	.	Mr E. M. Perry.
<i>Second Prize £3</i>	.	.	.	.	Mr J. Adamson.
<i>Third Prize £2</i>	.	.	.	.	Mr F. Hopkin.

CLASS PRIZES

Class D.

MEDICINE.—*First Prize*—F. W. Robards. *Second Prize*—J. Harrison. *First-Class Honour Certificates*—J. Adamson, E. P. Argyle, G. H. Barber, G. S. Bruce, H. Greenfield, L. G. Gryspeerdt, G. May, W. T. Olver, R. Paine, S. F. G. Pallin, E. M. Perry, W. E. Schofield, C. E. Turvill, E. C. Webb. *Second-Class Honour Certificates*—J. T. Angwin, G. H. Broad, J. R. Baxter, W. H. Chase, W. H. Edwards, H. H. S. George, A. W. B. Hodgson, F. Hopkin, H. H. S. Jones, H. D. Jones, E. S. Oliver, R. J. Sargent, A. G. Saunders.

SURGERY.—*First Prize*—F. W. Robards and E. C. Webb (æq.). *Second Prize*—G. S. Bruce. *Second-Class Honour Certificates*—J. Adamson, J. T. Angwin, E. P. Argyle, G. H. Barber, W. H. Chase, W. H. Edwards, H. Greenfield, H. H. S. George, L. G. Gryspeerdt, J. Harrison, A. W. B. Hodgson, F. Hopkin, G. May, E. S. Oliver, W. T. Olver, R. Paine, S. F. G. Pallin, E. M. Perry, R. J. Sargent, A. G. Saunders, W. E. Schofield, C. E. Turvill.

Class C.

PATHOLOGY.—*First Prize*—J. F. Craig. *Second Prize*—G. P. Male. *First-Class Honour Certificates*—J. R. Godbold, H. E. Gibbs. *Second-Class Honour Certificates*—A. Cornish-Bowden, S. Dodd, T. S. Green, A. Goodall, H. B. Hiles, W. Jurgensen, G. Sutton, C. W. Townsend, W. G. Wragg.

HYGIENE.—*First Prize*—G. P. Male. *Second Prize*—S. Dodd. *First-Class Honour Certificates*—J. F. Craig, H. E. Gibbs, H. B. Hiles, C. W. Townsend. *Second-Class Honour Certificates*—A. Cornish-Bowden, T. S. Green, A. Goodall, J. R. Godbold, W. N. Jurgensen, W. L. Little, T. Love, H. Newton, G. Sutton, P. G. Short, C. H. Sheather, W. G. Wragg.

MATERIA MEDICA.—*First Prize*—C. W. Townsend. *Second Prize*—H. E. Gibb and S. Dodd (æq.). *First-Class Honour Certificates*—J. F. Craig, G. P. Male. *Second-Class Honour Certificates*—A. Cornish-Bowden, T. S. Green, A. Goodall, J. R. Godbold, H. B. Hiles, W. N. Jurgenson, F. O. Parsons, G. Sutton, P. G. Short, H. Turnbull, W. G. Wragg, A. H. Watson.

Class B.

ANATOMY.—*First Prize*—A. S. Leese. *Second Prize*—H. Mason. *Second-class Honour Certificates*—W. Cawthorne, G. A. Dibben, A. N. Foster, R. J. Hills, F. C. O'Rorke.

HISTOLOGY.—*First Prize*—A. S. Leese. *Second Prize*—J. C. Broad. *First-class Honour Certificates*—T. S. Hunter, E. O'Reilly. *Second-class Honour Certificates*—T. Bowett, W. Cawthorn, G. A. Dibben, J. M'Rae Frost, A. N. Foster, R. C. Glover, H. J. Holness, R. J. Hills, A. R. Lewis, H. P. Lewis, H. Mason, F. C. O'Rorke, H. K. Roberts, F. R. Staples, W. H. Walker.

PHYSIOLOGY.—*First Prize*—E. O'Reilly. *Second Prize*—A. N. Foster. *Second-class Honour Certificates*—J. C. Broad, T. E. Burrridge, T. Bowett, W. Cawthorn, E. T. Clemow, G. A. Dibben, J. M'Rae Frost, R. C. Glover, R. J. Hills, T. S. Hunter, A. S. Leese, A. R. Lewis, H. Mason, F. C. O'Rorke, W. H. Walker.

Class A.

CHEMISTRY AND TOXICOLOGY.—*First Prize*—E. Brown. *Second Prize*—T. C. Graves. *First-class Honour Certificates*—J. Blakeway, W. J. Dale, R. L. Phillips, W. Urquhart. *Second-class Honour Certificates*—J. J. Aveston, R. Bennett, L. E. W. Bevan, R. Casey, C. Coles, W. T. Collins, P. Carter,

J. R. Hodgkins, B. H. Lane, E. S. Martin, B. M'Guire, F. T. Prince, D. Quinlan, S. Speer, R. W. Taylor, W. Trigger, E. R. Woodcock.

PRACTICAL CHEMISTRY.—*First Prize*—T. C. Graves. *Second Prize*—W. Urquhart.

BIOLOGY.—*First Prize*—T. C. Graves. *Second Prize*—E. Brown. *First-class Honour Certificates*—R. Bennett, W. J. Dale, R. L. Phillips. *Second-class Honour Certificates*—J. J. Aveston, J. Blakeway, L. E. W. Bevan, R. Casey, J. Hodgkins, D. Quinlan, W. Urquhart, C. W. Woodhouse, E. R. Woodcock.

MINOR ANATOMY.—*First Prize*—E. Brown. *Second Prize*—T. C. Graves. *Second-class Honour Certificates*—R. Bennett, L. E. W. Bevan, R. Casey, W. J. Dale, J. R. Hodgkins, F. T. Prince, R. L. Phillips, D. Quinlan, S. Speer, W. Urquhart.

PASS LIST.

The following are the Pass Lists¹ of this Institution for Session 1900-1901.

FIRST PROFESSIONAL EXAMINATION.

Messrs A. C. Anderson, S. G. Balls, H. C. Dibben, *T. A. Nicholas, *G. O. Ogden, P. J. Bolton, W. T. Christie, H. K. Roberts, J. Richardson, H. Sproston, *J. J. Aveston, B. B. Brown, *R. Bennett, E. C. Bate, J. Blakeway, *L. E. M. Bevan, †E. Brown, J. A. Bosley, *R. Casey, W. T. Collins, W. J. Dale, *T. C. Graves, *J. R. Hodgkins, B. H. Lane, A. Munro, E. S. Martin, R. H. Meade, B. M'Guire, S. H. Nixon, H. Platt, *D. Quinlan, F. V. Steward, *W. Urquhart, †R. L. Phillips, S. Speer, W. Trigger, *E. R. H. Woodcock, C. W. Woodhouse.

SECOND PROFESSIONAL EXAMINATION.

Messrs T. F. Hancock, W. L. Little, T. Love, H. Newton, F. D. Neal, A. V. W. Sewell, A. J. Thompson, W. F. Wilson, J. C. Broad, *T. Bowett, †W. Cawthorn, G. A. Dibben, A. N. Foster, †H. J. Holness, *R. J. Hills, †T. S. Hunter, *A. S. Leese, A. R. Lewis, H. P. Lewis, †H. Mason, †E. O'Reilly, †F. C. O'Rorke, F. R. Staples, V. de V. Woodley, W. H. Walker.

THIRD PROFESSIONAL EXAMINATION.

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¹ In this and the succeeding Pass Lists † indicates with First-Class Honours, and * with Second-Class Honours.

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DISPLACEMENTS OF THE BOWELS.¹

By J. R. U. DEWAR, F.R.C.V.S., Principal of the Royal (Dick)
Veterinary College, Edinburgh.

WHEN I promised that I would come to Newcastle and read a paper before the North of England Veterinary Medical Association, I did so without deciding as to the subject that was to be dealt with. After thinking the matter over for some time, and recognising that Newcastle was a busy manufacturing town requiring a large number of heavy horses to convey the raw materials and manufactured articles to and from its docks, wharves, and railway stations, I intimated to the secretary that I would deal with some affections of the bowels. There is little doubt that acute bowel diseases are more common in heavy draught horses than in harness and lighter bred ones. Still, there was a wide range of subjects, and I hesitated to begin. But an article which appeared in a professional journal not three weeks ago assisted me in coming to a decision. It purported to be the narration of a successful case, and was entitled "Obstruction of the Rectum: successful reduction of a twisted colon." That article gave me a title for my paper, or at least a subject to talk about. That will be "Displacements of the Bowels," though it will not be an attempt to deal with the subject fully, but will be mostly confined to cases I have met with in practice, and reflections on the etiology of the lesions.

In the December number of the *Journal of Comparative Pathology and Therapeutics* for 1891, the translation of an article by Professor

¹ A paper read at a meeting of the North of England Veterinary Medical Association, November 1901.

Möller of Berlin was published, on "Torsion of the left half of the Colon in the Horse." This was followed in the same number by a translation of an article on the same subject by Dr Malkmus, of Guben. The latter article had in addition to the same title as the first one,—“and its treatment by Retroversion.” This was followed in the issue of the same journal for the month of June following by an article, entitled “Malpositions of the Colon,” by the late Professor Walley. Since that date very little has appeared in our English journals on the subject.

It was, therefore, with considerable interest that I turned to the report of the case I have referred to. Surely after all those years of waiting here was now some fruit born of these articles previously mentioned. But the result was disappointing. The case bore a great air of unreality about it. Here was a mare which had been ill throughout the day, and by nine o'clock at night seemed hopeless. She was wandering round the box, sweating profusely, mucous membranes much injected, pulse very thready, and eyes amaurotic. It was found that she had an obstruction in the rectum, which was successfully overcome. It had obstructed the passage of one pellet of fæces; otherwise the rectum was practically empty. But this success did not cure the patient. The pain continued, and repeated attempts were made to reduce a right torsion of the left half of the colon, but without effect. At nine o'clock next morning another attempt was made to reduce this twisted colon, and this time successfully. How it was accomplished is not very clearly explained. But the practitioner is to be congratulated on the successful termination of a case which seemed to be *in extremis* twelve hours before the removal of the cause of the illness—the reduction of the twisted bowel—gave relief.

I must confess that I have not been so successful in cases of torsion of the colon, and I have had my own share of them. Some recover, some end in rupture, but more die from pain and exhaustion.

It is exceedingly difficult to explain satisfactorily how the torsion occurs, how the bowels become twisted, and the cause of the peculiar and puzzling positions in which they are often found. So recently as June of last year I made a *post-mortem* examination in a case which had run much the usual course. The animal had lived about thirty-six hours after the first appearance of illness,—longer than the average, but it all depends on the nature of the displacement. In this case the carcase was placed on its back, the abdomen opened, and the lateral walls reflected before the bowels altered their position. The sigmoid flexure of the colon was then pulled out from under the ensiform cartilage. That is to say, had the animal been alive and standing in its natural position, the sigmoid flexure, which normally should be about the brim of the pelvis, towards the upper hypogastric region, was resting on the ensiform cartilage, on the very floor of the epigastric region.

In this case, although there was no complete strangulation of the blood vessels, there was such a general congested condition of the organ as left no doubt as to the cause of death.

Another case met with earlier in the year was opened at the *post-mortem* in the same way, and a most impossible-like position of the organs was revealed. On the removal of the abdominal walls the

great mass of the colon, the first and fourth portions, was exposed at once. But the disposition of the parts was exceedingly abnormal. The origin of the cæcum and colon was so far forward in the abdomen that it seemed impossible for these organs to be in the position they occupied without some rupture or laceration of their attachment in the right lumbar region, but examination failed to reveal any laceration of the peritoneum. Instead of taking a course downwards and forwards, their course was downwards and backwards against the floor of the abdomen. The suprasternal and diaphragmatic flexures were practically effaced, and the sigmoid flexure, with the greater part of the second and third portions of the colon, had to be pulled out of the pelvis. The double colon, instead of being flexed on itself in its middle, formed a long wide curve. In this case death took place more rapidly than in the former one, although the appearance of strangulation was not much more distinctly marked.

Within a few weeks of the occurrence of this case, I had to make a *post-mortem* on a very different one. The horse was an average-sized cart gelding, rather lean, but in hard-working condition. At no time did he manifest symptoms of very acute pain, although these were worst for ten or twelve hours after the first appearance of illness. He lived a week after that, occasionally passing a little fæces, and at times partaking of some sloppy food.

All the time I was at a loss for a definite diagnosis. The *post-mortem* revealed a torsion of the small bowel; the ileum was twisted and constricted close to its termination at the cæcum. Although the constriction did not altogether obliterate the lumen of the bowel, yet the bowel in front of the constriction was considerably dilated. There was no distinct inflammation, but the twisted constricted portion was red and congested, though not much darker than port-wine colour. The stomach in this case manifested the most distinct hour-glass contraction I have met with in the horse, but I do not think it had anything to do with the horse's illness. Both these organs—the stomach and the twisted portion of the ileum—were preserved, and are now in the College Museum.

Subsequent to these cases, on the 29th September 1900, I made a *post-mortem* in a case which had surprised and disappointed me. The horse had been ill for nearly forty-eight hours on the last occasion I saw him. His pulse about 70 and fairly strong, temperature just under 103° F., and respirations moderate, were by no means alarming, and I had good hopes of his recovery. That was in the evening. There was all the time of his illness some movement of the bowels to be heard. Yet this horse, without manifesting any marked increase in the severity of the symptoms, at least to the day attendants, until just before his death, died between 5 and 6 o'clock the following morning. On making a *post-mortem* I was at a loss to make out at first what was the abnormality existing. The profession is not well situated for a *post-mortem* room in Edinburgh. It is with a grudge that the Sanitary Authorities permit us to open the carcases of animals so far, without spoiling the hide, as to enable us to make an examination of the abdominal and thoracic viscera. If we require to examine the central nervous system we must drive out to Loanhead, some 5 or 6 miles south of Edinburgh. So there is not the best chance of obtaining a satisfactory *post-mortem*.

In this case, on opening the abdomen and exposing the abdominal viscera there was nothing apparent so distinctly abnormal as to account for death. There was some congestion of the bowels, but not very marked, nor very extensive. The knacker was about to pull the bowels out, but I stopped him at once, and, stripping my arms up over the elbows, set to work to handle the viscera. It soon became evident that the small bowel was drawn together and constricted behind the liver and just under the spinal column. As the carcass was on its back and only the floor of the abdomen reflected, it was with difficulty that the constricted portion could be exposed; but with a little trouble this was done. It was then seen that part of the small bowel had passed through an opening in a direction from right to left and in the position indicated. The bowel was very little constricted, and not very much discoloured, and without the slightest difficulty some 5 or 6 yards of it were pulled back through the opening, passing from left to right. There was really no stricture and no obstruction to the circulation, yet though the bowel was not compressed, and slipped through the opening with the greatest ease, it had evidently caused the horse's death. After it was all pulled out, the opening—the foramen of Winslow—was found to readily admit two fingers, and was quite smooth, without any rupture of the peritoneum permitting of its enlargement.

I was surprised when I learned that this horse was dead, and the surprise was not diminished when I made out the peculiarity of the lesion, the absence of inflammation, of distinct strangulation, and even of very marked congestion.

These are only a few peculiar displacements, perhaps worth putting on record. So recently as the 8th of last month I made a *post-mortem* in a case in which the horse ate his breakfast and went out to his work in the morning apparently all right. He was brought in a few hours later evidently very ill. Soon after 9 o'clock I was called in to see him, and found him practically hopeless. I returned about noon and made the *post-mortem*. There was a twist of the colon, but the cause of the rapid death was an extensive rupture, fully 18 inches long, in the fourth portion, through which a great amount of ingesta had escaped into the peritoneal cavity—shovelfuls of it.

Although I have related two peculiar cases of displacement of the small intestine, there is little doubt that the most common abnormality of position in connection with the small bowel is when it is twisted round the mesenteric arteries as an axis. When the whole of the small intestine is twisted, when the strangulation is sufficient to practically arrest the circulation, death takes place in from four to eight hours.

It is as certain that the most common displacement of the large intestine is torsion of the left half of the colon, and my experience agrees with that of Jelkman, that it is generally twisted towards the right, *i.e.*, the upper or third portion is displaced towards the other half and downwards. But although torsion of the left half is most common, the twist and strangulation occurring at the suprasternal and diaphragmatic flexures, yet it may occur at any part of the free portion. I have several times met with cases where the twist was at the attachment of the organ in the lumbar region, and in these death took place in from four to six hours.

Etiology.—The cause of these displacements of the bowels is by no means clear. One reason for their greater frequency in heavy horses compared with light ones is probably to be found in the nature and quantity of the food. The heavy horse, as a rule, is accustomed to coarser and more bulky food, and the bowels are more voluminous, more capacious even comparatively. The abdomen, as a whole, is therefore larger. There is greater strain on the abdominal muscles of the heavy horse in starting a heavy load than in those of the light horse in moving himself rapidly. Greater care is also taken not to ask the light horse to exert himself severely with a full stomach and abdomen; very little attention is paid to this with the slow heavy draught horse. Much greater pressure can be brought to bear on the abdominal viscera when the abdomen is large and the line of the abdominal muscles considerably curved, than when the abdomen is small and the muscles acting on a nearly straight line. Although this easily accounts for hernias, the bowels, when exposed to great pressure, tending to escape in the direction of least resistance, it does not so readily explain those displacements that take place within the abdominal cavity. In the articles already referred to—translations from the German—the displacements of the colon seem to be mainly ascribed to sudden lateral movements of the animal. It is pointed out that, suppose a horse to be lying on his right side, the third portion of the colon is filled with more solid and heavier ingesta than the second, and that if he were to rise suddenly, it is quite conceivable that the third portion might slip down between the second portion and the fourth and first portions, and that if the second portion was much lighter there might be some difficulty in these parts regaining their normal position. But this does not explain those extensive backward and forward displacements which are to be met with, nor does it explain twists of the colon which extend beyond the suprasternal and diaphragmatic flexures. Yet in these latter, or even once a twist begins on the colon at its pelvic flexure, there is a possibility of it running to its lumbar attachment.

However, I feel convinced that in a normal condition, with the abdominal viscera only moderately filled, and the vermicular motion actively going on, were an accidental displacement to occur, the natural movement of the bowels would soon cause their return to their normal position; and that it is only when the organs are too full, unequally filled, subjected to severe strain, and severe pressure from the abdominal muscles, that displacement is likely to occur. In that condition, when the viscera are too full, or unequally filled, the abnormal position may be sufficient to arrest or modify the vermicular motion, so that there is little tendency for the bowels to return to their normal position.

This takes us to the second point, viz., that, after all, the great majority of these displacements are due to errors in dieting. There is no doubt that colic cases, stomach and bowel affections, are far less common since more care has been given to the feeding of horses, since less boiled bulky food has been used, which horses, as a rule, swallowed much too rapidly, and since horse-owners began to have a better appreciation of a rational method of feeding and watering.

That these dietetic errors lead to colic there is no doubt, and once a horse begins to roll and dash himself about, no one need be surprised

at displacements of the large bowels. The first and fourth portions are generally very much the heavier, and cannot but displace the lighter bowels when a horse dashes himself about, or rolls on his back. Then, if owing to inflammation or any other cause, there is any interference with the nervous stimulus, and the vermicular motion is arrested, it is not surprising if each successive tumble and roll displaces the bowel farther in the same direction.

I made a *post-mortem* in a very instructive case only this week. The horse showed some symptoms of colic one morning. The horse-keeper gave him an anodyne draught; he apparently recovered all right, and was sent to his work. In the evening the symptoms of pain returned, and he was treated with anodynes, purgatives, enemata, and hot fomentations. He settled through the morning, and remained practically quiet until about 7 P.M., when symptoms of acute pain returned, from which he never got any relief until he died about midnight. The *post-mortem* revealed a fairly acute gastritis of the pyloric portion of the stomach—not the whole, but about one third of it; an appearance of enteritis of the large bowels, the cæcum being darkest in colour, about claret colour, and the third portion of the colon least discoloured; and there was a partial twist of both cæcum and colon. When the abdomen was opened, and the abdominal walls reflected, the first and fourth portions of the colon were exposed; the other portions were partly hidden by the cæcum and small intestines, but it was the third portion that came first into view, not the second. And the second and third portions were to the right—had the horse been standing—of the first and fourth, but higher in the abdomen. That is to say, the cæcum and colon had together undergone a half twist. As already mentioned, the cæcum showed the most distinct appearance of strangulation. The practically fluid contents were evidently mixed with blood, the walls, in some parts, from a half to three-quarters of an inch thick, and blood infiltrated into their coats. In the colon this appearance was most distinct close to its attachment, and it got gradually less discoloured towards the pelvic flexure, save that the third portion was less discoloured than the second.

Now there can be no doubt that this displacement was not there when the horse was first observed showing symptoms of pain, more than forty hours before his death. Whether the pain had been due to the gastritis or to some other cause, I am not prepared to say. As already mentioned, the torsion had disguised any appearance of enteritis that might have existed in the large bowels. Nor do I believe that the torsion existed through the first night, the greater part of which he was manifesting pain; otherwise he would not have continued quiet for some twelve hours throughout the day. Possibly the action of the purgative on an already inflamed stomach and, it may be, bowel, had created some pain, which caused the horse to roll, and the displacement took place. From that moment he never obtained any relief.

There is here, then, an almost practical demonstration, that a twist may take place when an animal is rolling in pain; and an indication to the practitioner of the necessity for arresting that pain as soon as possible. But rolling will not account for those cases in which a horse feeds all right and goes out to his work to all appearance in perfect

health, and in the course of an hour or two is seized with an attack of violent pain while in harness, is with difficulty got home or into some convenient place, and never gets any relief until death. I cannot help thinking that, in these cases, the unequal fulness of the bowels, often with somewhat coarse food, and the great pressure they are subjected to when the horse is straining to start a heavy load, are accountable for the displacement.

Diagnosis.—As to our methods of diagnosis in cases of twists and displacements, they can only be characterised as most unsatisfactory. Although it is now nearly ten years since those articles I referred to appeared in English veterinary literature, I must say that I have never once been satisfactorily convinced in my own mind of the direction or character of a twist or displacement of the bowels during life. Yet in these articles it was referred to as if it were as easily made out as the presentation of a foetus in the uterus after the os has dilated. Perhaps I have been unfortunate in meeting with difficult or unusual cases, or it may be that my manipulative and tactile abilities are of a low order. But in the majority of the cases I have been quite unable to make out the pelvic flexure of the colon at all. It is quite different from manipulating a healthy horse with the organs in the normal position.

I have relied mostly on the acute, almost continuous pain; on the rapidly increasing frequency of the pulse; the almost entire absence of any sound indicating movement of the bowels; and, when the colon is extensively twisted, very often tympany. In addition, as a rule, there can be made out, on examination per rectum, a tense band, referred to by Jelkman, passing downwards from the lumbar region towards the floor of the abdomen, pressure on which causes pain.

Treatment.—The mechanical treatment of these cases has been in my hands perfectly useless. It could not well be otherwise when a definite diagnosis cannot be made. But even were a satisfactory diagnosis made, although only the left half of the colon is twisted, *i.e.*, the second and third portions, if there is a complete twist at the suprasternal and diaphragmatic flexures sufficient to cause strangulation, it seems absolutely impossible that any man could reduce it by manipulation with his hand in the rectum. At anyrate, if these portions are moderately full of ingesta, and the animal is one of our heavy horses, it could not be done. If only the posterior half of these portions is twisted, *i.e.*, about as much as is connected by the racket-shaped web of mesentery, it might be done. Several years ago, after lecturing on this subject to my students, one of them suggested to me, quite modestly, that he thought I was too sceptical regarding the possibility of reducing a twist of the colon in the living animal. Only a short time after, I took him with me to see a *post-mortem* in which I expected torsion of the colon. The carcass was turned on its back, the abdomen opened, the lateral walls reflected, and there was the torsion complete and quite evident at the suprasternal and diaphragmatic flexures. I asked him to try and reduce it. Although he was a man of good physique, he completely failed. He might have torn pieces out of the walls of it, but to turn it without taking it practically out of the abdomen, he was quite unable.

Of course, in the living animal, at least before there is much inflammation present, the tissues are softer and more pliable, the surfaces

smoother, better lubricated, and they slip easier on each other. But I would have much more faith in rolling the horse to effect reduction if an accurate diagnosis could be made. But that seems to me, as a rule, an insuperable difficulty. I have heard of a man who could examine the abdominal cavity so thoroughly that he was able to reduce a diaphragmatic hernia with his hand up the rectum. I rather think that was in America. But, although I have at least an average length of arm, I cannot claim such a marvellous power of abdominal exploration. And, as already mentioned, I never once, in the living animal, managed to satisfy myself, without any doubt, of the direction of a twist of the colon.

Regarding the medicinal treatment to be adopted in these cases, I am sorry that I have nothing new or very encouraging to bring forward. Certainly no uniform treatment can be adopted, and one must be guided by the conditions met with in each particular case. Anodynes are always needed. I am mainly guided in the administration of purgatives by the pulse being fairly strong and not very frequent, the mucous membrane not much injected, and the temperature not very high, with some intermission in the paroxysms of pain, and an indication of a full quantity of ingesta in the abdominal viscera on physical examination. For tympany I sometimes use eserine, occasionally with success, sometimes without. But if a full dose does not give the desired relief there is little use of repeating it. Some state that it should never be given without giving a dose of pilocarpine about ten minutes before it to increase the intestinal secretions. I have no experience of giving them in that way, and have often seen good results from eserine alone. But it is a very expensive drug, and I don't know any medicine in common use regarding which more care is required to obtain it thoroughly good and reliable. I have been unfortunate enough to use what I trusted was a solution of it when I might have as well used distilled water. Turpentine is a very useful if old-fashioned remedy in tympany. If the tympany is in itself sufficient to be causing distress I never hesitate to tap. There cannot be much chance of the reduction of a torsion if tympany exists and the abdominal cavity is packed full, and if there is any chance it is certainly much greater where the tympany is relieved and the tension relaxed.

There is really no risk in tapping with a proper trocar and ordinary antiseptic precautions. There is neither time nor convenience for shaving the spot; sometimes it is clipped, sometimes not. But it should be soaked with a strong reliable antiseptic, and the skin pressed firmly down to the abdominal wall while the canula is being withdrawn.

I have tapped, or rather inserted the trocar, in the right upper flank without any gas escaping, although the bowels were tympanitic, and on inserting a long probe through the canula withdrawn it moist but without a single speck of blood on it. I had simply passed between contiguous folds of bowel. I have tapped repeatedly on one side, I have tapped on both sides, and only once saw the slightest evil result from it. That was a case occurring in the country some twenty years ago, which had to be tapped with an old cattle trocar, or left to die. As it was, he ultimately died from a localised suppurative peritonitis. And I have tapped the same horse repeatedly. I met an old friend

at his work to-day,¹ a seventeen-hands lorry horse, which has been tapped at least half-a-dozen times and during several different attacks. At a *post-mortem* I made some years ago in a case of twist, I was very much surprised to find that the third portion of the colon had been tapped—punctured by the trocar—from the right upper flank, although it was almost at the other side of the abdomen on the *post-mortem* being made. That case was made clearer to me at the *post-mortem* I made this week than ever it was before. In this case the third portion of the colon was certainly nearer the right upper flank than the first or second, and in the former case it must have been the same when the horse was tapped, but the horse living a few hours longer, instead of becoming reduced, the twist had become more pronounced, until the third portion was again turned away from the right side.

I have never attempted to roll a horse for torsion, but could I satisfy myself as to the direction of the twist I would not hesitate a moment. It would not be always successful even were the diagnosis correct and the horse rolled in the proper direction. But it would have reason to recommend it. A full colon is not easily turned, and it might require some violence, or rather some sharpness in the turning, before it could be made to alter its position in the abdomen. Just as in torsion of the uterus, in which cautious turning is usually recommended, I have oftener than once found turning of no use until I used sharp, sudden jerks. After all they are nothing to the jerks a horse gives himself in dashing about, or the sharp, spasmodic act by which a horse while lying flat on his side will sometimes attempt to throw himself on his back.

Experience has shown me that there is a real risk of a horse producing twist while rolling in colic, and it has also shown that a horse may reduce a twist by rolling. I have repeatedly seen cases in which a horse after rolling almost in agony has risen and not shown any more symptoms of acute pain. So, once conviction is brought home to my mind that a twist exists, I prefer to allow a horse freedom to roll, and I believe that had horses larger boxes or paddocks to roll in, once they are affected with twist, there would be fewer deaths from that cause.

CONTAGIOUS PUSTULAR DERMATITIS OF SHEEP.

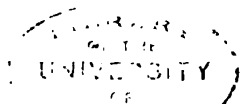
By ARTHUR H. BERRY, F.R.C.V.S., London.

General.

CONTAGIOUS pustular dermatitis, although perhaps more familiar to the shepherd than to the veterinarian, has, under the name of contagious dermatitis, or orf, in sheep, been the subject of attention by the late Professor Walley, by the late Professor Williams, under the name of *crusta labialis*, by the late Professor Robertson, under the name of carbuncle of the coronary band, and by Mr Clark, F.R.C.V.S., of Coupar Angus. It has also been designated "hair and hoof disease," and "mouth and foot disease."

The disease has been seen from time to time in many parts of

¹ 13th October 1901.



England, Scotland, and Wales. It more often occurs in low-lying, undrained, and wet lands, especially during or after prolonged or heavy rains, and where the herbage of such pastures is long, coarse, and inferior in quality.

Sheep of any age are affected, but it is frequently, and perhaps more readily, transmitted, and more severe, amongst lambs or sheep under one year old, many of the older sheep appearing to offer some resistance to the invasion of the contagium.

In many outbreaks nearly all the sheep become affected, and fresh sheep brought on with this disease will transmit the same to many other sheep that may be grazing alongside, the disease usually appearing within a fortnight after the date of mixing the flocks.

The fatality of the disease is not great, although fluctuating from time to time according to the wetness of the season. Those sheep which succumb are generally attacked with profuse diarrhoea, but whether this symptom is the outcome of the surroundings, such as climate and wet pastures, or due directly to the presence of this disease, I am unable to state. No doubt sheep affected with the disease are more easily brought under the influence of adverse surroundings than healthy sheep.

Symptoms and Lesions.

A catarrhal discharge from one or both nostrils or eyes may or may not be present.

The chief seats of disease are the nostrils, lips, and coronets, the malady appearing for the most part as a pustular inflammatory eruption of the skin of these parts. The lips and coronets are visibly swollen, and lameness (generally of one foot) is present amongst some of the affected sheep.

The lesions in the early stage exist as an acute inflammation of the papillæ of the skin, mainly confined to the parts previously mentioned. A simple lesion might be well termed in plain language a pimple (primary stage)—hard, scaly, and from the size of a small pea upwards. This pimple-like formation is attended with much inflammatory swelling of the skin, and localised, raised, and prominent papillæ are disseminated over its surface, with in a large number of cases a distinct and decided tendency to the formation of pustules.

The hair immediately in contact with the undisturbed lesion is glued together by the adhesive nature of the purulent contents of the pustule or small boil; it stands erect in small isolated patches or tufts, and is raised from the surrounding skin by the swelling of the pustule, together with the accumulation of proliferated epithelial cells upon its surface, which, to the naked eye, forms a hard scaly crust, constituting the scab, and varying in size with that of the lesion itself.

Upon pressure being applied (by manipulating the parts between the finger and thumb) to a well-developed and matured lesion, pus appears around the circumference and under surface of the scab, and when this scab or crust is rubbed off or removed it exposes an ulcerative and suppurating wound or sore, with (in some sheep) little or no tendency to heal naturally, but rather to the formation of irregular, soft, and unhealthy-looking granulations (which readily bleed) around the edges of the lesion. Suppuration occurs in many

instances to a greater or lesser extent from the interior of the sore. The wound itself has most frequently a well-marked circular margin, and when irregular, that is generally from the coalescence of closely adjacent pustules.

The chief seat of the lesions of the mouth is the outside of the lips, the upper lip being more often implicated than the lower. In many instances the pustules extend close up to the line of junction between the mucous surface and the skin, but with little or no inclination to extend or affect the mucous membrane proper of the mouth, although many small apparent pustules (like a pimple), of a shot-like consistence, in a certain number of cases, can be detected upon this line of junction.

Although the mucous membrane of the inside of the lips, cheeks, pad, or tongue is but rarely affected, or the seat of any apparent lesion, it is most important to recognise and to be prepared to find (in a small percentage of the most aggravated cases) the mucous membrane of the lips and tongue the seat of ulceration, and this appears to be entirely due to the discharge of a thin, ichorous, purulent fluid, plus an accumulation of effete matter around the commissures of the lips, coming in direct contact with these parts, and is quite a complication of the disease itself.

In the average case the lesions of the lips have less tendency to well-marked suppuration, they being mainly in the form of small raised pustules, rather numerous, with a natural tendency to coalesce, and giving the whole a moist, eczematous appearance.

There is no indication of a true vesicle, but it is always advisable to bear in mind the possibility of such from the application of too strong local dressings, such as paraffin, turpentine, strong acids, or other caustics.

I have in mind several instances where the owner (a farmer) had applied too strong local dressings, and in these cases the whole mouth and nostrils were much swollen and painful; vesicles or blister-like elevations were also present around the nostrils and on the inside and outside of the lips, which when burst allowed a thin serous fluid to escape.

In not a few animals the formation of pustules extends over the skin covering the nasal bones and around the orbit. Small pustules are also scattered upon the surface of the cheeks, about the size of a pea; and in these situations, as soon as the necrosed part becomes removed, the raw spot-like surface heals, or through inspissation of pus particles its development is arrested. It is not unusual to find in a small number of animals well-marked developing and matured pustules affecting the skin immediately surrounding the nostrils, which often, from excessive inflammatory swelling of these parts and consequent constriction of the nostrils, materially affects the process of respiration, the breathing being laboured and the ingress and egress of air accompanied by a noise.

In other animals isolated pustules may be so large as to constitute a fairly extensive abscess, affecting either the upper or lower lip, or both.

In aggravated cases affecting the lips the free surface and commissures show extensive disease, this being especially seen in those cases unattended with any attempt at local cleansing of the parts. The

opposing surfaces of the lips are a mass of hard, brittle, scaly, and necrosed crust, with much swelling affecting part or the entire length of one or both lips, and varying from half an inch to $1\frac{1}{2}$ inches or more in thickness.

There are present deep fissures or cracks running transversely across the scaly surface and extending well into the underlying structures. The same conditions are observable at the angle formed by the junction of the upper and lower lip, the upper lip being generally more extensively affected.

When the crust is removed by manipulation, it exposes a large, raw, angry, hæmorrhagic, and ulcerated surface, with specks of pus appearing at many points when the lip is squeezed between the finger and thumb. The fissures are still present, although, naturally, after the removal of the large crust, they are not so deep in appearance.

The accumulation of effete matter (saliva, particles of food, flies, dirt, purulent discharge, etc.) on the free border of the lips, and especially the commissures, has an excoriating effect upon the surrounding parts. The hair together with the epidermis peels off, as after applications of strong counter-irritants, leaving exposed a raw, tender surface, which in turn comes under the influence of any purulent discharge that may be present, or may extend into and implicate the mucous membrane and surface of the lips or tongue.

The opposite and corresponding part of the lower lip in some cases shows well-marked ulceration, although to a lesser degree, and it may be hidden from view by the swollen and inflamed overhanging upper lip, but is easily detected when opening the mouth.

Again, the formation of distinct pustules on the skin of the lower lip is often entirely absent, indicating that the part was most probably affected by direct contact with the extensively ulcerated surface opposed to it.

It is in these cases that the mucous membrane of the tongue may become affected, and thus, together with the ulcerated condition of the lips described, gives rise to a most offensively fetid smell, and a general appearance in the interior of the mouth like that of "ulcerative stomatitis"—an affection often met with among the smaller breeds of dogs from the presence of decaying teeth.

The ulcerative lesions of the tongue are not, as a rule, extensive; they occur on the side of the tongue that is nearest to the most aggravated lesions of the lips, and very rarely affect both lateral aspects of this organ.

The first appearance of this molecular necrosis is as a small superficial wound or sore, in which the piece of lost mucous membrane looks as if it had been "punched out," and the edges of the wound, that is, the mucous surface, are not free, but closely in contact with the underlying structures of the tongue.

The usual situation is a little in front of the first molar tooth, upon the supero-lateral aspect of the organ, with a tendency to extend in very aggravated cases to the edge and under surface.

The average size of the lesion is from that of a threepenny piece to a shilling, circular, or oval in outline, and the latter from 1 inch to 2 inches in length; but the size entirely depends upon the extent of the ulcerative and suppurative surface that the tongue is brought into contact with.

In these cases mastication during rumination appears to be entirely unilateral, the sheep performing the function with caution and care to guard the sores, and the condition gives rise to a little extra salivation.

The lesions of the feet are similar to those seen in the case of the lips, but often more aggravated.

The chief seats are the anterior and lateral aspects of the coronets of either the fore or hind feet, and, as a rule, the lesion is confined to these situations, although not always.

Some of the pustules, often as large as boils and abscesses the size of a medium-sized walnut, are in close connection with the coronary band; and these large pustules which have reached suppuration (with necrosis of the adjacent skin) cause a separation of the horn and skin, giving the appearance of a deep suppurating wound, like a small "quittor." If the cause is not removed, and there is persistent aggravation from the presence of dirt and an accumulation of purulent discharge which works its way downward under the horny laminae, the claw may be cast; but this is very rare unless the case is complicated with foot-rot.

Foot-rot commonly commences in the interdigital space, near the supero-lateral border of the horn, and extends downwards under the solar surface of the foot and upwards under the antero-lateral horn of the foot, the separation of horn taking place from below upwards. Foot-rot affects the horn proper, and is associated with the formation of large fungoid growths and a most offensive smell.

To resume,—the pustules are by no means confined to the coronets, but appear in many instances as high up the legs as the knees and hocks, sparing in number over the cannon and shank bones, but more numerous around the lower parts of the knee and hock joints respectively, and favouring the flexures of these joints.

The fetlock joints are also frequently and similarly affected, and occasionally extensive ulceration of the skin is met with. In many sheep well-developed pustules appear in close relationship to the supernumerary digits or ergot on all sides. The formation of a hard, painful, boil-like swelling is frequently met with here, apparently originating close under the base of the supernumerary digit, and with peripheral acute inflammation of skin and subcutaneous tissues, extending well into the hollow of the heel.

It is quite an occasional and uncommon occurrence to find any lesion affecting the soft part of the foot, that is, the heels or between the digits; consequently there is no separation of horn and skin at these particular parts, unless through extension of disease from pre-existing lesions affecting other parts. The lesions which appear on the front and sides of the coronets show in some of the more advanced and aggravated cases real abscesses or abscess cavities with thickened walls, accompanied by acute inflammation of the surrounding structures, and giving rise to lameness during locomotion, varying in degree in accordance with the size and extent of the lesions. Naturally those lesions nearest to the coronary band, affecting a part of extreme and delicate sensibility, and those aggravated by dirt or coarse, long, wet grasses (when the affected sheep stand holding the leg from the ground), are characterised by the most marked lameness; whereas less advanced cases and those of a more superficial nature are not associated with any observable lameness.

Differential Diagnosis.

As compared with contagious foot-and-mouth disease, the most characteristic features are that the disease is pustular, and not vesicular; that it affects the skin proper, and not the mucous membrane of the mouth, unless from unusual complications, and then the lesion is ulcerative, never vesicular.

The soft parts of the foot, *i.e.*, the heels and interdigital space, are but rarely affected, except from extension of disease; and the lesions of the foot in their early stage are always associated with pus, which is not the case in contagious foot-and-mouth disease. Separation of horn is not frequent. Cattle, although in direct contact, remain unaffected, whereas in contagious foot-and-mouth disease they never escape infection. The gait and appearance of the animal during locomotion is not as if all feet were affected, for the acute lameness generally arises from lesions affecting one foot only.

The spread of the disease is not so rapid, emaciation is not well marked, sheep, on the whole, feed well, and the general appearance of dejection and ill-health is absent, except perhaps in a very few instances. There is no constant snatching and picking up of the fore limbs, or kicking out of the hind limbs. The sheep are not found lying scattered about the pastures as in contagious foot-and-mouth disease, or at most only a few individuals in a flock are thus separated from the others. Lameness is not well marked, but naturally it is increased with the severity and aggravation of certain cases; it is entirely absent in others. There is more evidence of pain from pressure than pain from irritation and soreness.

Treatment.

Complete isolation of the affected flock. Where possible, a change of pasture should be made, and the preference given to high, well-drained, and dry land. If practicable, a small quantity of dry fodder should be given morning and evening, such as hay, chaff, linseed and cotton cakes. The use of rock salt is not without recommendation. Medicinal treatment, if any, should consist of stimulants, alteratives, and tonics; a mixture of common salt, iron, and gentian added to the fodder will be found very useful.

Local treatment is attended with well-marked results; and if the sheep are taken in hand before the lesions become aggravated by dirt and the presence of accumulated discharge, they speedily recover.

In those cases where the scabs are very hard, and cannot be separated with ease, application of an antiseptic ointment will facilitate their removal.

In the average case the appropriate local treatment consists in a thorough cleansing of the parts in water, removal of scabs and all effete matter, and rubbing the legs with the hands while each foot is placed in a shallow bucket or trough containing a solution of some antiseptic, such as lysol, Jeyes' fluid, carbolic acid, Condy's fluid, or hyposulphite of soda. One or two thorough dressings to the affected parts are generally sufficient, but the worse cases will require attention until improvement is observed. It is most astonishing how speedily the majority of the sheep recover after one thorough dressing.

THE SCHMIDT TREATMENT FOR PARTURIENT PARALYSIS.¹

By JOHN J. REPP, V.M.D., Veterinarian to the Experiment Station, Ames, Ia.

THIS disease is also known by the names parturient paresis, paresis of parturition, parturient fever, milk fever. The word "fever" in connection with the terminology of this disease is not very appropriate, because in the majority of cases fever is not present, but the animal has a subnormal temperature. The term milk fever is very misleading and indefinite, as it is also used by the laity to designate other diseases, such as parturient septicæmia and the various forms of mammitis. Parturient paralysis must be clearly differentiated from parturient septicæmia, which is a disease of an entirely different character and which may occur in any of the domestic species, whereas parturient paralysis occurs only in the cow.

History.—Although this history of the disease is rather obscure, it is probably quite an old one.

Distribution.—Parturient paralysis occurs wherever milk cows are kept. It is more prevalent in dairy districts, because it is the heavy milking strains of cows that are most subject to the disease. As far as I am able to learn, it is quite prevalent in certain sections of our own State, while in other sections it rarely occurs. In the same locality more cases occur in some years than in others. This irregularity of distribution does not appear to throw any light upon the cause of the disease.

Cause.—No definite cause can be assigned for this disease. We cannot do more than theorise upon this point. The scope of this article will not permit the discussion of these various theories, therefore it shall be limited to a brief consideration of the theory which more especially concerns the present discussion—namely, that of Veterinarian J. Schmidt, of Kolding, Denmark. Schmidt's theory is that parturient paralysis is caused by the evolution in the mammary gland of a poisonous substance through the over-activity of the epithelial cells of this gland excited by the determination to the udder after birth of large quantities of blood which was supplied to the uterus and the foetus before birth, but which now goes to the udder because of the natural demand for milk secretion. This poisonous substance being carried in the circulation to various parts of the body, brings on the symptoms which characterise the disease. It is well recognised that living cells may, under certain circumstances, produce poisonous substances. Schmidt's theory, therefore, is in accord with an established principle.

Pathogenesis, or Generation, of the Disease.—Parturient paralysis, as a rule, occurs in cows which give a heavy flow of milk and which are in a high state of nutrition. It may develop at any age, but is extremely rare in cows before they have reached adult age and have given birth to several calves. It is also rare in old cows. It occurs, then, in cows which are of middle age and in the full height of their

¹ Reprinted from the "Journal of Comparative Medicine and Veterinary Archives," September 1901.

activity as milk producers. The disease attacks the cow after she has given birth to a calf, usually within twenty-four hours thereafter, but in some cases not until a week or even a month after parturition. In a few cases the disease has its inception a short time before parturition. Cows which are stabled and deprived of exercise are said to be more prone to the disease than those which are permitted to exercise at will. There are many exceptions to this statement, although it is the usual teaching. Further observation may show that it is not correct. The report given in this bulletin indicates that in Iowa more cows take this disease while at pasture than in any other circumstance. This doubtless arises from the fact that in Iowa cows are given more freedom than is customary in older dairy States. The disease may arise at any time in the year, but, on account of the fact that spring-time is pre-eminently the calving season, most cases originate at this season.

Morbid Anatomy.—The morbid alterations are limited and variable, and offer nothing characteristic. The blood is irregularly distributed, a condition which probably indicates marked vasomotor disturbance resulting from the profound interference with the nervous functions which accompanies the disease. The abdominal organs are usually filled with blood. The brain may be anæmic, œdematous, easily torn, and yellowish in colour. In other cases it shows hyperæmia of the meninges and of the brain substance.

Symptoms.—The disease usually appears within twenty-four to forty-eight hours after parturition. In extreme cases it may not occur until two months or even six months after parturition. It may rarely occur before birth. It usually follows an easy birth. At the onset of the disease the cow manifests some uneasiness; she moves about in a restless manner, stamps, strikes the abdomen with hind legs, perhaps bellows, grinds the teeth, and may have spasms of groups of muscles or even a general convulsion. After this period, which may be unnoticed, the symptoms of paralysis come on. The cow shows weakness, staggers, and at last falls. As the paralysis advances she stretches on the ground, lying on her side usually with the neck bent to one side so as to bring the nose into the flank or the costal region. This is the characteristic position of parturient paralysis. If the head is brought into the normal position, it at once returns to the unnatural position in which it was found. The animal is in a state of partial or complete unconsciousness, does not respond to blows or calls, and takes no note of her surroundings. The eye is dull and not sensitive to the finger touch, sunken, pupil dilated, and the upper lid is drooping; the tongue is paralysed, saliva runs from the mouth, the pharynx and œsophagus have lost the power of motion, so that the animal is unable to swallow; the peristalsis of the stomachs and intestines is in abeyance, and as a result digestion is arrested, fermentation sets in, and the animal becomes tympanitic; the contents of the rectum and colon are hard and dry, and may be covered with mucus or blood, urination is suspended; the os uteri is almost invariably dilated if the disease occurs within a day of parturition; pulse small, often imperceptible, 60 to 120 per minute; temperature usually normal or below normal, may be as low as 95° F., in some cases may be as high as 105° F. Such a high temperature probably does not occur in a case of pure parturient paralysis, but

only when there is a complication of parturient septicæmia. The extremities are cold. The after-birth is sometimes retained. There may be accompanying prolapse of the uterus.

Course.—Without treatment, and, indeed, with most kinds of treatment which have been applied in the past, the disease usually runs rapidly to a fatal issue. It lasts two to three days, and in some cases longer, the condition gradually becoming more and more aggravated. Death results from sudden failure of the heart or brain, and is often preceded by profuse diarrhœa. In milder cases the cow may linger as long as two to four weeks and then die of pneumonia, which results from the inhalation, or introduction through attempts at medication, of foreign substances into the lungs during the period of paralysis of the pharynx and œsophagus. If recovery occurs, the animal is entirely well in two to five days. In rare cases paralysis of the hind parts may persist for a long while.

Diagnosis.—This is made by a study of the history and symptoms. It is comparatively easy.

Differential Diagnosis.—It must be distinguished from antepartum paralysis, broken-back, parturient septicæmia; but one familiar with the character of these diseases will find no difficulty in making this differentiation.

Treatment.—This may be considered under two distinct subdivisions, viz.: preventive treatment and curative treatment.

(a) Preventive treatment.—By considering what has been said under the head of "generation of the disease," one can easily infer what measures should be adopted to prevent the disease. Cows in the later stage of gestation should be fed moderately, grain especially being given sparingly or entirely withheld; the animals should be given an opportunity to take plenty of exercise; the bowels should be kept in good condition by the administration of such salines as magnesium sulphate, sodium chloride, and sodium bicarbonate. The afterbirth should be removed soon after parturition and several uterine douches administered.

(b) Curative treatment.—The object of this bulletin being to report the results of the treatment of parturient paralysis by the method first suggested and recommended by Schmidt-Kolding, the discussion shall be confined to that treatment to the exclusion of the many others that have at various times been brought forward and tried, but which give but little encouragement for the continuance of their use.

Schmidt does not claim that his method of treatment disposes bodily of the morbid condition, but that it does measurably assist nature in her efforts to restore the animal to the normal physiological state. It is well known that after the beginning of the attack the animal, if left to herself, rapidly grows worse until the crisis of the disease is reached, at which time death occurs or convalescence begins, usually the former. It has been observed, however, that if the treatment is applied within a few hours after the inception of the disease its progress is modified in such a way that convalescence at once begins, as a rule, and the animal hastily recovers her health, usually within twelve hours, although in extreme cases it may be as late as forty-eight hours. Following is an outline of the plan of treatment of parturient paralysis suggested by Schmidt. The operator should disinfect his hands and the udder and teats of the cow by

washing with a 5 per cent. solution of carbolic acid or creolin, or a 1½ per cent. solution of lysol or trikresol. The apparatus needed for the treatment consists in a small glass funnel, a rubber hose three feet long and one-eighth inch in calibre into which the funnel fits, and an ordinary milking tube over which the rubber hose fits. This apparatus should be sterilized immediately before it is used by boiling or soaking in such a solution as recommended for washing the udder. Dissolve from two to two and one-half drachms of potassium iodide—the size of the dose depending upon the size of the cow and the character of the attack—in about one quart of clean water previously boiled to sterilize it, and allow the solution to cool to a little above body temperature, or 40° C. or 104° F. The temperature may be determined with the clinical thermometer. Withdraw all the colostrum or milk from the udder. Then insert the milking tube, with hose and funnel attached, into one of the teats, elevate the funnel about two feet above the teat and slowly pour in one-fourth of the solution, allowing the funnel and hose to become empty several times during the process in order to permit the entrance of a liberal quantity of air. Repeat this infusion with the other three quarters of the udder. After all is introduced knead the udder carefully so as to cause the solution to permeate the ducts and acini as much as possible.

As the condition of the cow is usually such as to indicate additional treatment, the veterinarian should not be content with simply the infusion of the potassium iodide solution, but should resort to other measures which promise assistance.

As the cow is usually unable to urinate, the bladder will be found filled with urine. This should be removed with the catheter, and its removal accomplished at intervals until the recovery of the cow renders this procedure no longer needful.

It may be advisable that catharsis be brought about. As the cow is usually unable to swallow, it is dangerous to attempt to give medicines by the mouth. This may be done if assurance that the cow can swallow is obtained. Some have given medicines successfully through a probang inserted into the stomach. This plan is feasible. Schmidt says that he usually resorted to an aloe powder. If this is done one ounce to one and one-half ounces of aloes may be given. It would seem preferable to give the aloes in a bolus, capsule, or drench. Some have given linseed oil or Epsom salts. If the animal cannot swallow and a probang is not at hand, one may administer one and one-half to two grains of physostigmine salicylate subcutaneously, repeating the dose in about three hours if purgation is not produced. Rectal injections should be given at short intervals in order to get rid of the accumulation of hard, dry feces in the rectum. These injections may be of linseed oil, cottonseed oil, or warm soap solution. Schmidt recommends, also, enemata of sodium chloride solution. Meanwhile the cow should be kept propped up on the sternum by means of bags of straw or pieces of wood. If the temperature is below normal, as it usually is, the cow should be thickly clothed with blankets and straw heaped up about her. Schmidt used powdered digitalis given by the mouth when the heart was rapid and weak. It would seem much better in every way to give the tincture of digitalis subcutaneously. He has also resorted to subcutaneous injections of camphor and caffeine. This is good treatment. If the cow does not show marked

improvement within eight hours the potassium iodide infusion may be repeated. Schmidt has found that as high as six drachms may be given into the udder without harm to the cow. Schmidt, in his first report, made in 1898, recorded fifty cases treated for parturient paralysis by this method with but two deaths from the disease. There were, however, only forty-six recoveries, as two cows were slaughtered for beef during the first day of convalescence. A short time later a report was made by Jensen showing that in Denmark up to that time sixty-five veterinarians of that country had treated 412 cases by the Schmidt method, 90 per cent. of which recovered. These results seem to indicate that this was the treatment *par excellence* for this disease. It still remained to secure the introduction of this treatment into the United States and to determine what results could be obtained by our veterinarians through this application. For the purpose of assisting in the introduction of the Schmidt treatment for parturient paralysis into Iowa, and of determining what the results of this treatment would be in the hands of the veterinarians of this country, in September 1899 I addressed a circular letter to each of the 150 graduate veterinarians in Iowa requesting their co-operation in the work of this research by applying the treatment in their practice and reporting their results to me. Ninety of these replied, signifying their willingness to use the treatment. I should say that a few of these had already put the treatment into use in their practice. These, however, agreed to report their subsequent results to me. Accordingly, a circular of instructions, an infusion apparatus, two ounces of potassium iodide, and ten special blanks for report of cases treated were forwarded to each veterinarian who had consented to co-operate. In November 1900 a letter was issued to each of these veterinarians requesting that the reports they had collected be sent in. Up to this time I have received replies from thirty-three veterinarians. In all 166 cases were reported; of these 166, 119 resulted in recovery, while 47 were fatal. Of the fatal cases, in 8 of the cows death may be traced to some complication, such as prolapse of the uterus, foreign body pneumonia, etc. In these cases the Schmidt treatment cannot be said to have failed, for it is not in any way intended that it shall be able to overcome such accidental conditions. If the cow has recovered from her condition of paralysis as a result of the Schmidt treatment far enough to be out of danger from that source and to promise recovery, but later falls a victim to some complication that is in no measure a part of parturient paralysis, but only a result of that disease, it may with justice be said that the Schmidt treatment was a success so far as the malady against which it was directed is concerned. Looking at the reports from this generous point of view, in 127 cases out of 166, or 76.5 per cent., the Schmidt treatment was successful so far as the parturient paralysis was concerned.

I regret that the limit of this article forbids the presentation of a full report of each case, and that it must be confined to the following tabulated report, which gives a summary of the important features of these reports:—

[TABLE.

1. Breed—			
Shorthorn	131	Galloway	1
Jersey	17	Polled Angus	1
Holstein	4	Devon	1
Hereford	3	Grade	42
Not stated	66		
2. Age—			
Four years	2	Nine years	10
Five years	8	Ten years	8
Six years	19	Twelve years	5
Seven years	24	Thirteen years	1
Eight years	26	Not stated	63
3. Condition—			
Fat	33	Thin	2
Fair	57	Not stated	74
4. Quantity of milk given—			
Very large	41	Small	6
Large	24	Not stated	95
5. Care and feeding before parturition—			
Fed lightly	3	Pasture, other feed	19
Stabled, fed liberally	14	Not stated	78
Pasture only	52		
6. Had parturient paralysis before—			
Once	4	Twice	2
7. Character of parturition—			
Normal	80	Not stated	83
Difficult	3		
8. Number of calves borne by each cow—			
One calf	1	Six calves	14
Two calves	2	Seven calves	9
Three calves	13	Eight calves	2
Four calves	19	Nine calves	1
Five calves	19	Not stated	88
9. After-birth retained—8.			
10. Prolapsus uteri—2.			
11. State of os uteri during attack—			
Relaxed	28	Not stated	130
Contracted	8		
12. Disease appeared before parturition—1.			
13. Length of time between parturition and first symptoms of disease—			
One hour	2	Twenty hours	9
Two hours	1	Twenty-four hours	19
Three hours	2	Thirty hours	8
Six hours	2	Thirty-six hours	9
Eight hours	7	Two days	5
Ten hours	6	Three days	7
Twelve hours	9	Five days	1
Fourteen hours	7	Eight days	1
Sixteen hours	5	Not stated	59
Eighteen hours	7		

Time between inception of disease and beginning of Schmidt treatment:
One to twenty-four hours.

¹ The figures represent the number of cases.

14. Number of times infusion of potassium iodide was repeated—
 Once 22 Twice 2
15. Time elapsing between application of Schmidt treatment and time the cow was able to stand—
- | | | | |
|--------------------------|----|-----------------------------|----|
| One hour | 1 | Sixteen hours | 6 |
| Two hours | 1 | Twenty hours | 5 |
| Four hours | 1 | Twenty-four hours | 4 |
| Five hours | 1 | Thirty hours | 5 |
| Six hours | 11 | Two days | 6 |
| Eight hours | 10 | Three days | 2 |
| Ten hours | 5 | Four days | 3 |
| Twelve hours | 5 | Not stated | 94 |
| Fourteen hours | 6 | | |
16. Number of relapses—4 (fatal).
17. Number of cases of pneumonia—6 (fatal).
18. Quantity of milk secreted during first five days after treatment—
 Small 26 Not stated 137
 Normal 3
19. Number of cases in which Schmidt treatment was followed by mastitis—7.

That in many cases in the above table certain features are put down as "not stated" is in large measure accounted for by the fact that only 110 of the reports were made on the blanks which I sent to the practitioners, the rest being made in the form of a brief summary in a letter. Of these reports made on the blanks only a few gave in detail all of the particulars called for by the questions. I infer, however, that the features set forth in the table may be taken as a fair representation of all the cases treated.

The table shows that most of the cows affected were grades. I may add that most of these were shorthorn grades. Next in point of numbers is the shorthorn breed. It should not be inferred from this that the shorthorn breed is more susceptible, for it is probable that the order of numbers affected among the various breeds is in close proportion with the number of cows of these breeds in the State.

It will be seen by reference to the table that cows affected were chiefly between the ages of six and nine years, and at the birth of the third to the sixth calf. This is in accordance with observations elsewhere. Only one of these cases occurred in the primipara, and only two in the secundipara.

Nearly all the animals were reported either fat or very fat. Only two cows affected by the disease are reported as thin in flesh. This also is in accord with the usual observation.

The table is in a measure contradictory to some of the current teachings on this subject in respect to the care and feeding of the animal prior to parturition. In the majority of the cases in which this feature was reported upon, the cows were at pasture and had no other food. They also had plenty of exercise. Only fourteen were stabled and fed liberally, conditions generally thought to predispose to this disease. Only nineteen were at pasture and received in addition other food. That most of the cows took the disease while at pasture and not receiving any other food may arise from the fact that this is the way in which most cows are kept in Iowa during the

pasture season. We cannot conclude that pasture alone may not constitute a liberal diet to a cow with good appetite and digestion, although it is generally considered that if the grass diet is not supplemented with grain ration the cow is reasonably safe from an attack of parturient paralysis. The above figures would indicate that the conclusion in this respect needs some revision. It is encouraging to note that only three cases occurred in cows fed lightly with a view to preventing the disease.

It is worthy of note as being in accord with the usual teaching that nearly all the cows were large or very large milkers.

In nearly every case the parturient act was accomplished in a normal manner. In only three cases did parturient paralysis develop after a difficult parturition. This, however, may be in the same ratio as the normal to the difficult births not followed by the disease. Were this so it would not damage Schmidt's theory of the etiology of the disease, for in order to prove his theory it is not at all necessary to show that the disease followed an easy birth, although Schmidt cites this circumstance in support of his theory.

The table would indicate that one may look for this disease most commonly within the first twenty-four hours after the calf is born. In one case, however, it had its onset before parturition; in others, after the first day.

The table shows that the infusion of potassium iodide into the udder was repeated in the course of eight to twelve hours in twenty-two cases, and was repeated twice in two cases. Of the twenty-two cases which received two doses each, seven were fatal, two having had relapses and one pneumonia. There is no means of learning from the reports just what the second or the third dose contributed toward the cure of the disease, but as all these cases were severe ones and the repetition of the dose was strongly indicated, it may be inferred that it is good practice to repeat the dose in eight to twelve hours, provided the cow has not then responded to previous treatment. I think it may be safely concluded that twenty or even thirty grammes of potassium iodide injected into the udder within twenty-four hours will not do harm, but, on the other hand, may contribute largely toward the cure of the case. It would not be advisable, however, to repeat the dose if the cow has responded to the first one.

It appears from the table that one may expect the cow to have pretty well recovered from her attack within twelve hours after the treatment is administered, although some cases do not yield so soon. A relapse may be expected at any time, and it would seem that this renders the prognosis very grave.

Pneumonia is a very grave complication. None of the six cows in which this complication arose recovered. This leads to an inquiry into the cause of the pneumonia. It may be due to dust or particles inhaled during the attack, but it is most likely set up in most cases by the entrance into the lungs of drenches designed for the stomach, but failing to reach their destination on account of the inability to swallow. This would lead me to recommend that no medicines be given by the mouth while the animal is unable to swallow, unless given by means of the probang. The heart stimulants, such as strychnine, atropine, caffeine, tincture of digitalis, can readily be given hypodermically, as can also the cathartic in the shape of a

dose of physostigmine. One would judge, however, that, since physostigmine is such a powerful motor depressant, it would be contraindicated in this disease, yet it has been given with favourable results. For my own part I should not use it, and cannot recommend its use. It is not manifest upon examining the theory of the etiology of the disease and the state of the alimentary canal that a cathartic is strongly indicated. The fecal matter is usually normal except in the lower part of the large intestine and the rectum, from which places it may be dislodged by enemata and by manual means. Would it not be well, then, to withhold the purgative at least until the cow has regained the power to swallow? I believe, withal, it is good practice to give a cathartic whenever the cow is able to swallow.

One practitioner who has treated a large number of cases with very good success frequently gives nothing but the infusion of potassium iodide into the udder. There is no doubt that many cases are hurried on to a fatal termination by excessive medication. Just where to draw the line is a difficult point which each practitioner must decide in the case before him.

The report of seven cases followed by mastitis warns us that too much care cannot be exercised in sterilising everything that comes into contact with the udder during the process of introducing the potassium iodide solution. It is certainly true that the potassium iodide alone will not give rise to this accident, which should not be charged against the treatment. It is probably the result of infection introduced into the udder by lack of adequate disinfection.

In most cases reported milk secretion was much below normal in amount for a few days after the injection of the potassium iodide, but was restored to normal in all cases in which mastitis did not develop. This is to be expected, for the treatment brings success partly because it diminishes cellular activity in the udder.

Although the reports do not show the relative value of treatment early in the disease and late in the disease, for some cows treated early promptly died, while others treated as late as twenty-four hours got well; yet it is fair to conclude that the earlier the treatment is instituted the more likely is it that the cow will recover.

In the event that the cow suffers from tympanites during the attack of parturient paralysis, resort must be had to puncture of the rumen with the trocar. Some veterinarians have administered medicine through the canula after the puncture has been made and the gases evacuated. This procedure is doubtless as useful as it is unique.

With 76.5 per cent. of cures to the credit of the Schmidt treatment in the hands of the general practitioner in Iowa, who is called upon to treat these cases under all sorts of adverse circumstances, it only remains to advise that this treatment be promptly resorted to in all cases of parturient paresis. If it is not infallible it is at least the best form of treatment for this malady that has thus far been introduced.

It is also to be expected that as veterinarians acquire more practice in its application they will be able to apply it better and with a greater degree of success.

TUBERCULOSIS A PARASITIC DISEASE, AND ANALOGIES.

A REPLY TO EDITORIAL CRITIQUE.

By THOS. B. GOODALL, F.R.C.V.S., Christchurch.

AFTER reading the remarks of our worthy Editor in his critique on my paper in the last issue of the *Journal of Comparative Pathology*, I felt very much as I should imagine a Gentile would, who had innocently stepped into the Holy of Holies (where, of course, he had no business to be), and there had the misfortune to tread on the toes of the High Priest, by whom he would be promptly anathematised.

Except from this point of view (the trespassing in subjects that are sacred to the select few), I am at a complete loss to understand the reason of the Editor's severe criticisms, and I can only attribute his outburst to a temporary aberration of the functions of a peculiarly talented mind.

I was, at first, inclined to let the article, with the critique, pass without comment, leaving it to anyone who might care to read them to judge of their merits, for I really have no motive, and nothing to gain, by entering now into a controversy with a public man.

But then again, no one with any feeling of self-respect could allow such severe criticisms to be made from such a source, without, at least, an endeavour to save himself from a position of ridicule.

The Editor's remarks demonstrate only too clearly what a narrow groove even the most gifted and talented of men may get into—their minds may become so set on one object that they will not see anything beyond that. In the case of bacteriologists, if we are to take this editorial article as an example, it appears that they will not allow the possibility of any factors except bacteria exercising any influence in the causation of health and disease. Such is their sense of proportion! Whereas the biologist notes cause and effect ever at work amongst the highest as well as the lowest forms of life, and a rational study of biology teaches that the bacteria are only one of many forms of beings, that they have their rôle in the scheme of life, like other organisms, and that the pathogenic bacteria exert their power and influence most when higher forms of life are not conforming to their environments, which is very much the case with our domesticated stock. They are a great power to be reckoned with, but not the only one, for general health and robustness count for much in resisting them.

We are warned against the fallacies of reasoning from analogy. Well, probably, no two enquiring individuals can see things in exactly the same light. To me it appears that reasoning by analogy and inductive reasoning have been the means of bringing to light almost everything that has ever been revealed. Of course, there have been and always will be fallacies. For instance, it is reasoning by analogy to infer that because we think we have stamped out a disease amongst cattle by slaughtering all affected and in-contact animals, therefore it is practically possible, by the same methods, to stamp out all

other diseases, and this sometimes leads us into the committal of very grave errors.

But it is generally safe in the consideration of such questions as I have chosen for the title of my paper to assume that "if two or more things resemble each other in many points, they will probably resemble each other in more points."

And if I find, as the result of observation, that almost every animal has its own parasites, which are generally not easily transmissible to another animal, except by artificial methods, and then not easily, and if we grant further that bacterial diseases are parasitic diseases, it cannot surely be fallacious to assume that bacterial diseases will probably resemble other parasitic diseases in this respect, until it has been proved experimentally that it is not so. This was my sole object in submitting my original paper for publication.

My argument is, that tubercle bacilli are parasites, and so far as we at present know, each animal has its own particular tubercle bacilli, not easily, by natural means, transmitted from animal to man. This is the argument by analogy; it has to be proved by the experiment-
alists whether this reasoning, generally true in other like things, holds good in the particular case under discussion.

Objection is taken to the use of the phrase "popular scientific," and we are told that it is not easy to understand what is meant when two terms that are generally used in opposition are compounded to form a new adjective. I wonder whether my critic has ever heard of a Popular Science series of books, edited by no less a personage than Lord Avebury, better known as Sir John Lubbock, as sound a scientist as any man, and withal a man of practical commonsense. It is no adjective of my own compounding, but has been in use in the English language for a number of years, and its meaning is pretty generally understood. Let us analyse this compound adjective.

I have some remote notion that "popular" has some connection with "people," and I believe that "people" are generally regarded as human beings—men and women.

What are scientists? All professional men are scientists, because they know certain things as facts, and the bulk of professional men are ordinary human beings, and it was to this class that I applied the term; but from the inference of my critic I should be led to judge that there are certain exceptions, that some scientists are "*sui generis*." They may not be classed with people, so where do they come in? Man was made a little lower than the angels, so that the expert scientist must be either higher than the angels, or lower than man in the scheme of life. Which is it?

The notes of my paper were prepared long before the meeting of the Congress in July, or perhaps the compound would not have been used, for when we read of a man (or rather a scientist) who injects poisonous material into the anterior chamber of the eye of a heifer, that he may watch the results, causing the eyeball to swell to twice its natural size; and when such experiments are published without shock or shame, without one word of comment, it reads to me like a case of "reversion to type," to barbarism in its most primitive and cruel form.

This may be scientific, but in this country, at any rate, it would not be popular, and I should be sorry for the scientist who would dare to publish an account of such experiments if our people got hold of him.

My experiments may be childish, but these are devilish and brutal ; and I must thank the Editor for having put me right on this point—that expert scientists of this type cannot be classified with the human beings of the ordinary kind.

Further on I am twitted with dishonesty of purpose in not mentioning more than a certain number of diseases as analogous to tuberculosis, and the Editor gives a list of diseases that I ought to have quoted if I had been honest ; he says that each of these in his list resembles tuberculosis in being transmissible to several of what are called the lower animals.

Let us remember that the argument of my paper is that tuberculosis is a parasitic disease, that every animal has its own particular parasites, which, as a rule, are peculiar to itself, and, judging from analogies, until it is proved to the contrary, tuberculosis might in this respect be placed in the same category as other parasitic diseases.

I made the experiment with the apples to prove that given sound skins and mucous membranes we should probably escape many bacterial diseases, which, for the sake of argument, I classify with other diseases due to parasites.

And my contention is that, given these conditions, these *are very rarely* indeed transmitted to man. I do not suggest that they are never conveyed, but that the occurrence is very rare.

Granting then these premises, I do not think a better list could have been chosen to give emphasis to the point of my argument than the one submitted by the Editor.

Rabies.—No better example could have been given than this, for it is probable that every dog bitten by a rabid dog contracts the disease, *but only a very small percentage of human beings that are bitten develop hydrophobia*, and then *only by inoculation*, so that, given a whole skin, that is, one not penetrated by the teeth of the dog, how many men would be infected ?

Glanders.—Very easy of transmission from horse to horse, but man is very rarely infected, and in these exceptional cases only by inoculation.

Anthrax.—I can scarcely agree with the Editor that there is much in common between anthrax and tuberculosis, but even in this disease it is generally by inoculation that man becomes affected, though when the spores are inhaled in large numbers, probably inducing violent expiratory efforts and causing slight injury to the mucous membrane, he may become infected in this way.

Actinomycosis.—I do not believe to be contagious in the ordinary acceptance of the term. In April 1890 I placed on record a case I had of a cow being badly affected for a whole year with open wounds, discharging the fungi freely, and this cow was running all the time with others, but not one of them contracted the disease. Neither animals nor man become affected with actinomycosis except by a wound of the skin or mucous membrane.

Ringworm.—I will concede this to the Editor, though even the transmissibility of this disease from animals to man is not nearly of so common occurrence as a few recently recorded cases would lead us to believe. Since I have been in practice I have treated probably several hundreds of cases in horses and cattle, and I can only remember some three or four occasions on which the attendants have contracted the disease.

When the Yeomanry horses were being drilled into shape here, some of them were literally smothered with ringworm, but I never heard of any of the men, who were really very careless, being affected.

So much, then, for the Editor's list, for which I proffer him my thanks.

Before leaving this point, I should be very sorry indeed to give a suspicion of an impression that I do not fully appreciate the danger to man of one and all of these diseases, and indeed, of tuberculosis as well, until it is proved to the contrary, as I have taken care to mention in my original paper. There is no man living more careful, either as regards himself, or in his solicitude for others who might be running the least risk of infection from any one of these diseases; and yet, I say, none of them is so easily contracted as is generally allowed.

In his reference to what I say about "cause," the Editor has given an entirely wrong meaning to my argument, and, to be honest, he must know that he has. He says I fall into the venerable error of attributing tuberculosis to some cause other than the bacilli. I do nothing of the sort, and he cannot twist my words to convey any meaning so utterly foolish. The very title of my paper disproves this, for I start with *the assumption* of tuberculosis being a parasitic disease; but I do strenuously maintain, notwithstanding the Editor's opinion to the contrary, that anything that tends to lower the vital energy of an animal renders it more prone to attack, and that, therefore, this primary lowering is a cause of attack. Surely that is plain enough, and a fact so simple and plain that I should have thought it could not have been controverted, except, as I said, through some temporary aberration of the mental faculties.

I notice that the Editor scornfully ignores hereditary predisposition as a factor. Well, we may go for analogies in this to man, and is it not only too painfully true that certain families have the hereditary weakness? I am honestly of opinion that this is so, and I notice, too, that in the tenth resolution passed at the recent Congress it is stated that the following question be submitted for the consideration of the next Congress on tuberculosis:—"The constitutional conditions of the individual which predispose to tuberculosis, and the means by which they may be modified."

Is not this intended to throw some light on the question of hereditary predisposition? It will need very astute arguments and very strange conclusions to prove that this is not a factor, and certainly in the present state of our knowledge everything points to the probability that it is.

My experience in testing herds has been the same as that of others alluded to by the Editor. Sometimes those *apparently* healthy and robust react, while wiry, hard, thin animals stand the test. But in determining what are healthy and what are not, the Editor has quite overlooked the fact that I am arguing from Nature's point of view, and from this standpoint much of the stock that we admire so much are weaklings and monstrosities, entirely dependent on artificial aid to hold their own in the struggle for existence, and if this aid were withheld for ever so short a time they would die off, the prey to pathogenic organisms, tubercle and others.

The Editor gravely assures us that it is a fact that there are

thousands of herds infected with tuberculosis where breeding from immature parents is not practised!

I would ask him, Is a bull mature at nine months old? which is the age at which he is often worked, particularly in ordinary dairies, and I would remind the Editor that we have not only to tackle the disease in highly-bred herds, but also amongst our common dairy cows. I have often had the curiosity to enquire into the reason of their being used so young, and the answer always is that a young bull is more manageable, he does not give so much trouble as an older one, and as long as he "stops" the cow that is all that is wanted.

But where are we to find the thousands of herds where the practice is not in vogue? At what age would the Editor consider cattle to be mature? It is the universal custom to breed from bulls at fourteen or fifteen months of age, and to breed from heifers at that same age. Are we to consider these mature?

If this is an example of the facts on which modern scientific deductions are founded, it is no wonder we find so many of them collapsing, and new facts being substituted every few years for old fallacies which were thought to be facts. The worst of it is that we generally have to accept facts as they are given us, because we have no means of controverting them, but in this particular case we can take the fact for what it is worth, and still form our own opinion.

I honestly believe that it is probable that the practically universal custom of breeding from immature parents, together with the crowding together of young calves, without fresh air, sunlight, and exercise, make the animals so bred and so brought up an easier prey to the tubercle bacilli than if more attention were paid to conformity to environment as insisted on by Nature.

We are advised that we need not trouble much about such minor considerations as general health and upbringing, but that we must only tackle the bacillus.

I must confess that my sense of proportion is so hopelessly defective (these are the Editor's words), that I attach very great importance to these considerations. I must go to analogies once more. Why do we insist on our young children being kept as much as possible in the fresh air and sunshine? Is it not, as we say, to build up their constitutions that they may be strong and vigorous? What for? Just that they may resist the attacks of pathogenic organisms, which we are just beginning to learn are ubiquitous, or nearly so.

Well, if experience has taught us that our children benefit and are more disease-resisting for such treatment, surely it is only reasonable to use the same methods for the upbringing of our young stock, that they also may be better able to resist. I would refer anyone interested in this subject to Professor Ewart's Penycuik experiments, which prove conclusively how necessary fresh air and sunlight are for the healthy breeding of young animals.

But we are told that the question of general health is venerable, old fashioned. Let it be so. I shall insist on instilling the principle on all whom I may be called upon to advise, until it can be proved to me that this is only of minor importance to the killing of the bacilli, and in the present state of our knowledge it would be the wisdom of members of the veterinary profession to so advise.

I must confess to a feeling of astonishment that anyone in the position of the Editor should gravely hint that, if called upon to advise how to combat tuberculosis, we might practically ignore the influence of breeding, or of the manner of housing young stock. He would say, "go on with your old methods as long as you like, only avoid contagion."

How does he reconcile this again with the finding of the London Congress, with regard to human tuberculosis, but which is quite as applicable to stock?

"That in the opinion of this Congress overcrowding, defective ventilation, damp and general insanitary conditions, diminish the chance of curing consumption, *and aid in predisposing to, and spreading the disease.*"

To sum up,—In dealing with tuberculosis it should be our endeavour to get rid of all clinically diseased and reacting stock, and in this both I and the Editor are agreed; and then, but here we appear to differ, to encourage the breeding, and bringing up under sound hygienic principles, of healthy stock, not paying so much attention to artificiality, as to more natural environments, and I believe that we could in this manner check the bacilli. I should not have the remotest fear for myself, for example, that, given my present healthy, out-of-door life, tubercle bacilli could make any impression on my body, though they might be about me.

It does not follow that, because in the dressing of surgical wounds we can prevent the ingress of pathogenic organisms, we are to conclude that they are all destroyed; no, they are very much in evidence, as we know to our cost if we are the least negligent in our treatment.

So with these bacilli, tubercle, and others; they are not all going to be destroyed just yet, but we should endeavour to make it as difficult as possible for them to attack successfully our cattle, and one way of doing this is by keeping the latter healthy and vigorous.

MALIGNANT JAUNDICE IN THE DOG.

By WILLIAM ROBERTSON, M.R.C.V.S., Government Veterinary Surgeon, Cape Colony.

THIS disease, variously named malarial fever, malignant malarial jaundice, and by some dog-owners simply distemper, is one of the most fatal diseases of the dog met with in Cape Colony.

It may be roughly defined as a malarial disease affecting dogs, communicable by direct inoculation from animal to animal, but not by cohabitation or ingestion, and due to the presence of a hæmatozoon in the red blood corpuscles.

The disease attacks Cape bred and imported dogs indiscriminately, is confined to certain parts of the country, and is most prevalent in the summer and autumn.

Judging by contemporary literature on the subject, there can be no doubt that this disease is met with affecting dogs in other countries. Celli, in his work on "Malaria," alludes to Pointer dogs brought from Lombardy to the Compagna marshes contracting a form of malaria, and mentions the presence of an intracorpuseular body in the red

blood corpuscles. In 1890 Piana and Galli Valerio pointed out in the blood of the dog in Italy an intraglobular hæmatozoon resembling in form the pyrosoma bigeminum of Smith and Kilborn. At the Société de Biologie, 20th January 1900, M. P. Leblanc of Lyons Veterinary College evidently describes a case of this disease under the heading "Nature of Infective Icterus in the Dog," as he alludes to a hæmatozoon analogous to that found in red water in the ox. Professor Koch has seen the double pyrosoma in the blood of dogs in East Africa, and Dr Marshon verified its existence in Senegal in the blood of eleven dogs. Lastly, in the *Recueil de Médecine Vétérinaire* (reprinted in the *Veterinary Journal*, 1901) Messrs Nocard and Almy in France describe a case which in clinical history, symptoms, and microscopical appearance of the blood, resembles a description of a good case of malignant jaundice of the dog in Cape Colony.

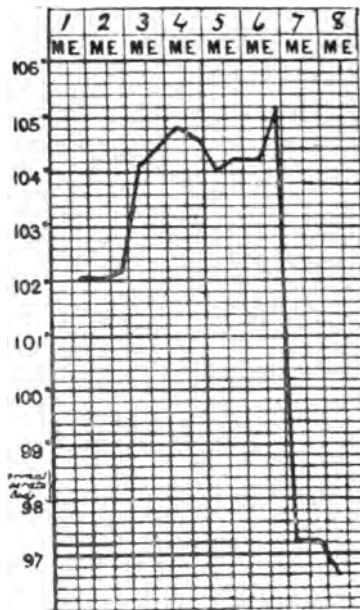


FIG. 1.

Temperature chart of a case of malignant jaundice in a dog inoculated intravenously with 5 cc. of blood (from a dog naturally infected).

Symptoms.—In a case produced by subcutaneous inoculation of a healthy animal with infective material, the temperature rises on the third day, and death supervenes on the twelfth or thirteenth. If, on the other hand, the inoculation is intravenous, the initial rise of temperature still occurs on the third day, but death takes place much sooner—from the eighth to the ninth day, or even earlier.

The first clinical symptoms are noticed on the third day after inoculation; the dog is dull, listless, drowsy, refuses food, and may be thirsty. On the fourth day in a case of intravenous inoculation you see the biliary staining of the visible mucous membranes commencing, just a faint dirty-yellow being noticeable. During the following day the characteristic hæmoglobinuria may make its appearance, the urine

being in many cases as dark as claret. All the visible mucous membranes are of an almost chrome-yellow colour, and any naked skin, such as flanks, prepuce, inner edge of ears, etc., shares in the staining. The animal lies perfectly helpless; the whole body exhales a foul odour, constipation is commonly present, the tongue is foul and furred, the teeth dirty, and there may be shedding of part of the gums down to the alveoli. The animal in most cases appears to die from collapse and weakness, due to the drain upon the blood produced by the hæmoglobinuria. That this is not *always* the cause of death I am sure. Just as in Texas fever (or red water as it is called in Cape Colony) one may get an animal dying with all the symptoms of that disease except the hæmoglobinuria, and find the typical pyrosoma in the blood, so one may get a dog with all the other symptoms of malignant jaundice except hæmoglobinuria, and, as in the case of the ox, recover the special pyrosoma from the blood.

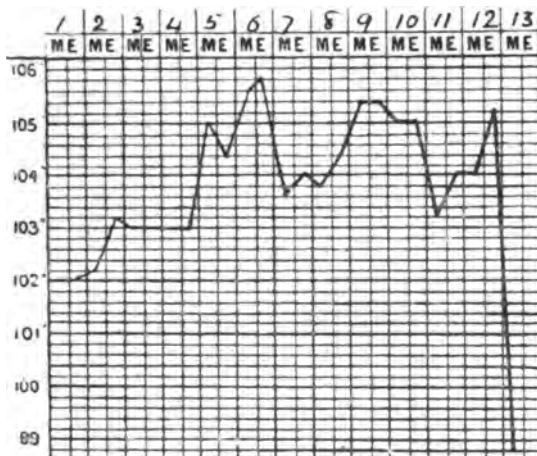


FIG. 2.

Temperature chart of a case of malignant jaundice in a dog inoculated subcutaneously with 5 cc. of blood (from a dog naturally infected).

If the case of jaundice has been produced by subcutaneous injection the course of the disease is slower, but the termination and the general train of symptoms are the same.

Post-mortem Appearances.—I take two cases at random from the *post-mortem* book.

CASE I. 20th October.—Subject—A small smooth-haired fox-terrier dog; died during night; inoculated on the 13th inst. with 5 cc. blood intravenously from a case of naturally contracted disease in a collie. Body emaciated, weight 10 lbs. 4 ozs.; eyes sunk in the head; visible mucous membranes and bare places on the body, such as the prepuce, flanks, etc., of a dirty-yellow colour; tongue heavily furred; teeth covered with dirty yellowish-brown deposit, possessing a most foetid odour; subcutaneous fat, subcutaneous connective-tissue, and superficial muscular tissue stained yellow; intraperitoneal fat ditto; heart shows petechiæ on epicardium and endocardium; liver much

enlarged, friable, yellowish-red in colour, weight 310 grammes ; spleen very much enlarged and distended, pulp much softer and darker than normal, weight 61 grammes ; stomach quite empty, congested at fundus, mucous membrane generally bile-stained ; small intestines bile-stained ; large intestines inflamed throughout, with copious exudate of a rusty-red colour on the mucous membrane, no ulcers visible ; kidneys externally bile-stained, and on section paler than normal in the cortex ; bladder contained about 50 cc. of dark claret-coloured urine, with a foetid odour. Microscopically the blood and spleen showed the presence of the characteristic pyrosoma.

CASE II. 17th October.—*Subject*—A small, smooth-haired, fox-terrier dog. This was a case of natural infection, first noticed sick on 7th October, was not treated during the course of the disease, and died on the morning of 17th October. Body in good condition, weight 10 lb ; all visible mucous membranes and skin of belly stained yellow ; subcutaneous tissues and fat of a bright yellow colour ; heart normal ; liver much congested and enlarged, more friable and

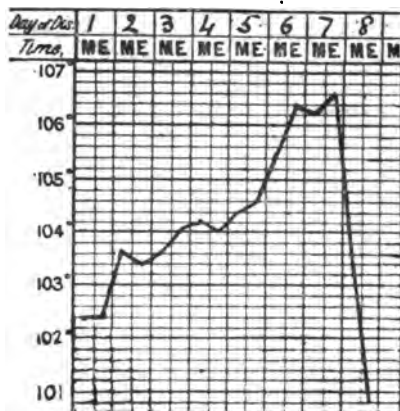


FIG. 3.

Temperature chart of a case of malignant jaundice in a dog inoculated intravenously with 5 cc. of blood (from a dog infected by inoculation).

darker in colour than normal ; spleen much enlarged and softened ; stomach had mucous membrane congested, and contained about an ounce of a dark coffee-coloured liquid ; small intestines normal ; the large contained slightly brown or rust-coloured mucus ; no ulcers anywhere ; kidneys normal ; bladder full of claret-coloured urine. Microscopically the blood showed the presence of a double pyrosoma in the red corpuscles.

From a study of the *post-mortem* records of these two cases, one naturally infected, the other produced by inoculation with infective material, it will be seen that the *post-mortem* appearances in the two cases were very similar. The most striking features in the *post-mortems* I have made are, the deep bile-staining of skin and mucous membrane, the enormous increase in size of the spleen, the blood-coloured urine, and the peculiar odour evolved from a living or freshly killed dog badly affected with the disease. The increase in size of the spleen is perhaps the most marked *post-mortem* lesion.

Causal Parasite.—This is without doubt a double pyrosoma, with, in the complete stage, the thin ends approximated, and resembling in its general appearances the pyrosoma bigeminum of Texas fever. This pyrosoma in the dog was first demonstrated in South Africa by Dr Carrington Purvis, at the Bacteriological Institute, Grahamstown. A few months afterwards Mr Spreull, M.R.C.V.S., one of the Government veterinary surgeons, proved the inoculability of the disease. He says, "Intravenous inoculation I found produced acute symptoms in about four days, and death about the sixth; subcutaneous inoculation, on the other hand, was much slower, death occurring from the eleventh to the thirteenth day, after two or three days' illness." Since then I have made extensive inoculation experiments and *post-mortem* examinations in connection with this disease. On one occasion I obtained blood from a sick dog, and kept up the disease through thirteen animals, every one of which succumbed.

I find the intracorpuseular bodies mentioned by Dr Purvis to be

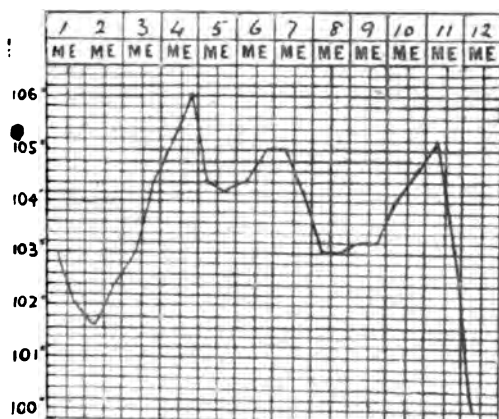


FIG. 4.

Temperature chart of a case of malignant jaundice in a dog inoculated subcutaneously with 5 cc. of blood (from a dog infected by inoculation).

present in every case of the disease met with. The blood is infective on the third day, and they make their appearance in the red corpuscles on the fourth day after subcutaneous inoculation with virulent blood. I have also found the pyrosomata free from the corpuscles in a very advanced stage of the disease.

The pyrosomata are met with in smears from liver and spleen, and the latter organ in a fatal case positively swarms with them, one corpuscle having as many as five or six bodies in its interior.

Though in many ways strongly resembling the pyrosoma bigeminum of Texas fever, the intracorpuseular bodies in malarial jaundice in the dog present certain well-marked differences. They are larger, and not so markedly pyriform as the Texas fever parasite, and present somewhat of an oat shape, staining slightly in the centre, which possesses one clear spot, and more deeply at two or three places in the margin. This parasite stains well by carbol thionin blue, methylene blue, and the double stains of Plehn and Czinz-is-kis, but

does not take up the hæmatoxylin when using Cole's method of eosin and hæmatoxylin. It is found in sections of tissues, and blocking the capillaries in the brain. In some cases single bodies in a corpuscle may be met with, and I do not think they appeared only when the blood was examined in the early stage of the disease, as I have single pyrosomata in specimens taken at *post-mortems*. Using the blood of a case which showed the pyrosoma microscopically, I was unable to infect the horse, sheep, ox, cat, fowl, guinea-pig, rabbit, rat, or mouse.

Therapeutic Remedies.—While keeping up the strain of blood for inoculation experiments, I had an opportunity of trying therapeutic remedies, with marked ill success, but I do think the disease was aggravated, and rendered more virulent, by passage through dogs. All sorts of remedies likely to have a beneficial effect were tried—quinine, calomel, ammon chlor, ext. belladonæ, carbolic acid, benzoate of soda, alone and in combinations, without very satisfactory results.

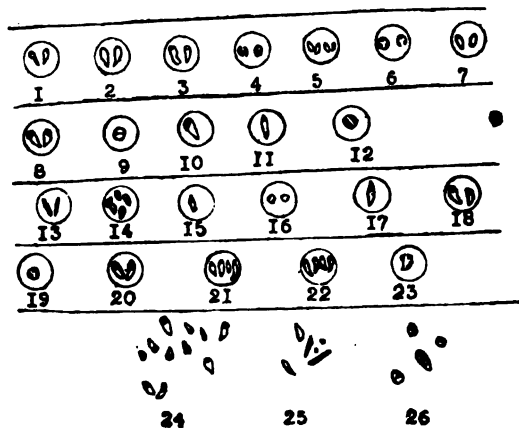


FIG. 5.

Various forms assumed by the causal parasite of malignant jaundice in the dog.

Nos. 2, 8, and 20 are the forms most commonly met with. Nos. 24, 25, and 26 are parasites free in the blood-stream.

I certainly think carbolic acid hastened death. The treatment giving best results was a calomel pill to start with, then a calomel and quinine pill four times a day (the calomel must be given in very large doses). Good nursing and careful diet are, of course, essential to a satisfactory termination of the illness.

Means of Infection.—Having settled the causal parasite of this disease, one naturally next desires to find out the bearer of this cause of infection. At present we have only the history of cases of natural infection and the analogy of the spread of another disease associated with a pyrosoma to guide us to any decision in the matter; and, judging from the history of many cases of malignant jaundice, I feel little doubt that the infection in this disease can also be conveyed by ticks. I will give a couple of cases of natural infection, as sort of circumstantial evidence.

CASE I. Subject—A small rough-haired mongrel dog, kept for the past two months in close confinement in a glass case, and used by Mr Chas. Lounsbury, the Government Entomologist, as a host for rearing certain ticks from various domesticated animals.

This dog became ill, and presented all the symptoms of malignant jaundice, with pigmentation of visible mucous membranes, and presence of pyrosomata in the blood. The dog eventually died, and the *post-mortem* appearances, macroscopical and microscopical, were typical of a case of that disease.

Mr Lounsbury tells me that some time before the dog fell sick he obtained from some dogs in the neighbourhood of Cape Town (which dogs were said to be salted or immune to malarial jaundice) a few dog ticks, and at the same place and time secured one tick from an animal affected with the disease. These ticks were hatched out in the Laboratory; during the process the tick from the sick dog got missed and the larval ticks placed for the purpose of feeding upon the rough-haired dog in the case. I feel certain that larvæ of the tick from the dog suffering from malignant jaundice got mixed with the larvæ of the salted ticks and so produced the disease, as we know larvæ from ticks off salted animals will not do so.

CASE II. Subject—A small aged Pointer dog, the property of a gentleman in the suburbs, who had kept this dog at his house for some two years.

History.—This dog, which, by the way, the owner said had had this sickness before, was taken on a shooting trip to Kuils, a very bad tick veldt, ten days before, returned with ticks on him, was first noticed sick three days ago, and had rapidly got worse.

When I saw the animal it was lying prostrate, unable to stand or move. Mucous membranes of eyes and mouth, the naked skin of the flanks, even the internal surface of the ears were stained a deep dirty yellow, and there was a drop of dark blood-coloured urine on the tip of the prepuce. The teeth were covered with sordes, and the tongue with a thick yellowish-green fur; the mouth had a most disagreeable odour.

I took blood from the jugular vein, and this under the microscope proved a most curious specimen, as coverglass after coverglass was examined without finding a complete red corpuscle, but every field teemed with pyrosomata free, some lying with their thin ends approximated, as seen in the red corpuscles, while others appeared singly or in groups of three or four. I stained with Plehn's and eosin, then methylene blue, and in all the covers not a single red corpuscle took the eosin stain, all that remained of them being some granular débris.

The dog lived twenty-seven hours after the abstraction of this sample of blood.

The inference I drew from the above case was as follows:—

The neighbourhood of the dog's home is not a tick veldt, and the animal is taken to a tick veldt, and to a neighbourhood where malignant jaundice is known to exist, returns with tick attached to his skin, and shortly after succumbs to this disease. These facts being so, I do not think that it is too much to say that the dog contracted the disease from the ticks at Kuils River.

In conclusion, I should like to point out that I have seen cases of

jaundice affecting dogs in England in which the mucous membranes and even the skin showed bile-staining, and I think it would be worth while for some of the members of our profession to make and examine a few coverslips of blood from the next case they meet.

I have attached to these notes four temperature charts, showing the characteristic temperature curve in this disease, whether produced by subcutaneous or intravenous infection. It will be noticed that the curve in a dog infected from a case of natural infection is the same as when the infecting material is taken from an artificially produced case.

THE PART PLAYED BY THE DOG TICK, "*HÆMAPHYSALIS LEACHI*,"
AS A CARRIER OF INFECTION IN MALIGNANT JAUNDICE OF
THE DOG.

The fact that certain external parasites can and do carry infection from a sick to a healthy animal is, I think, acknowledged by most authorities. Smith and Kilborn have shown that the progeny of ticks, *rhhipicephalus annulatus* (Say), act as carriers in the case of Texas fever or red water in cattle. The value of such knowledge is apparent, as by its use we are able to minimise the spread of such diseases, *e.g.*, we spray cattle to kill ticks before changing pasture, and prevent them carrying Texas fever to a clean area, and cover the pools with kerosene to destroy the young of the malaria-bearing mosquito.

The following experiments, conducted by Mr Charles Lounsbury, Government Entomologist, and myself, throw some light upon the dog tick as a carrier of infection in the disease known in this country as malignant jaundice of the dog. I have previously mentioned two cases of malignant jaundice, and the chain of evidence which prompted me to state that I considered the dog tick to be capable of conveying the contagion of that disease. It will now be seen from the experiments detailed below that artificially hatched ticks, the progeny of females matured on affected animals, can produce the disease in healthy dogs; but only when they reach the adult stage.

From specimens submitted to him, Professor G. Neumann, of the Veterinary School, Toulouse, identified the South African dog tick as *hæmaphysalis Leachi*; it was this tick which we used.

The brood ticks from which the eggs, and later the young ticks employed in the following experiments, were obtained, came from a naturally infected dog—a mongrel, alluded to in the previous notes, and quoted as a case in support of the tick-borne theory of the disease. These ticks laid eggs, which were hatched out on moist sand in an incubator. The dogs used for the first experiment were two fox terrier puppies, which from birth, and till the termination of the experiment, were kept in such isolation as to preclude the chance of natural infection. When ten days old, the dogs were placed under observation; temperatures were normal, and no parasites could be found in the blood. When about a month old, on 15th and 20th June, both puppies were infested with larval (newly hatched) ticks. The temperatures were now regularly taken, and the blood examined at frequent intervals, but no fever manifested itself, and no parasites could be found in the blood.

In July the same dogs were again infested with a batch of the same ticks, which had moulted and entered the nymph stage; again no result followed. On 27th August the same dogs were infested with a batch of the same ticks as before, but now in the adult stage. The dogs began to look sick on 9th September, temperature rose to 105° , and on examination of blood films the red corpuscles teemed with parasites, some corpuscles containing as many as eight of the pear-shaped forms. These intracorpuseular bodies varied in shape, some being spherical or ring shaped, with a darkly-staining wall, and some thickening of this, or condensation of protoplasm, at opposite sides of the circle; others were typical pyrosomata, two being the most frequent number noticed, but one, and up to eight, were also found in a single red corpuscle. Some parasites were found free outside the blood corpuscles, both spherical and pyriform in shape, and even clove-like, that is, the thickened portion becoming suddenly thinner, instead of tapering like an oat seed. Some of the intracorpuseular spherical forms, which had a deeply-stained portion at either side of their periphery, seemed to have a fine partition dividing them in two. The puppies died on the 14th and 15th days, respectively, after infection with the young fully-formed ticks.

Post-mortem appearances were those of typical malignant jaundice. Liver much enlarged and congested; spleen large, very soft, and dark in colour; blood thin, coagulating imperfectly; a few soft clots in the right ventricle, veins empty; subcutaneous tissues and peritoneal fat bile-stained; urine colour of claret and water; kidneys enlarged, pink in colour, rather firm, and on section glistening like paraffin wax.

I am certain:—

(1). That the dog from which the brood female ticks were taken in the first instance was suffering from the disease, as the microscopical examination of the blood during life showed numerous intracorpuseular bodies, and a *post-mortem* examination confirmed the diagnosis.

(2). That the hatched-out progeny from these brood ticks produced malignant jaundice in the two puppies, from which disease they died.

Why the puppies did not contract the disease when infested with the ticks in larval or nymph stages admits of two explanations:—either (a) very young dogs may be naturally immune, up to a certain stage, or (b) very young ticks from infected brood females are unable to produce the disease. To ascertain which was the correct explanation, I performed the two following experiments:—

(1). One of the sick puppies was bled at the height of the disease (temp. 105° F. and many parasites in the blood), and 3 cc. was injected into the jugular vein of a healthy twelve-months old dog on 9th September. Intracorpuseular bodies appeared in the blood on the 11th, on the 15th the temperature rose to 105° F., and the animal died from malignant jaundice on the 18th.

(2). A puppy fourteen days old (one of eight), sucking its mother, was also inoculated with a minute dose of the same blood on 9th September. Intracorpuseular bodies appeared in its blood on 14th September, temperature 103.2° , and death from malignant jaundice took place on 20th September.

The blood of both animals was examined previous to inoculation, and found to be free from pyrosomata.

As confirmatory evidence that dog ticks, the progeny of infected

females, can only convey the disease during the adult stage of their growth, a second series of experiments was undertaken. Dogs Nos. 5, 6, 7, and 8 were selected, and kept under such circumstances that natural infection was an impossibility; for some time previous to infesting them with ticks their temperatures were carefully registered and their blood frequently examined.

Dogs Nos. 5 and 7 were infested with larval ticks, the progeny of infected females. The temperatures were taken regularly, and no rise was noticed; films of their blood were taken at frequent intervals, and no parasites could be seen. These two dogs are alive and well at time of writing.

Dogs Nos. 6 and 8 (a puppy and a full-grown dog respectively) were infested with ticks in the adult stage, the progeny of infected females, on 23rd September. On the 4th October the temperature of No. 8 was noticed to be above normal. Films from the blood were at once examined, and the causal parasite of malignant jaundice found to be present in the red corpuscles. On 5th October the temperature of dog No. 6 showed a rise, and microscopical examination of the blood revealed the presence of the typical parasite.

That artificially-hatched adult ticks, the progeny of infected females, can produce this disease in healthy animals, I think must be admitted.

Age does not appear to give any protection from this disease, as in previous experiments I have been able to produce it by subcutaneous and intravenous inoculation in dogs of all ages, and with the utmost certainty.

In conclusion, it may be added that large quantities of dog's blood, containing the parasites in large numbers, have been injected intraperitoneally into guinea-pigs, pigeons, etc., without producing death, or even any lasting constitutional disturbance. This experiment was undertaken to ascertain if the parasite of malignant jaundice in the dog produced the caustic and irritant effects said to follow an intraperitoneal injection of Texas-fever blood, as stated by M. Lignière in his report on that disease in Argentina.

THE VETERINARY INSPECTION OF IMPORTED AMERICAN HORSES.

By PERCY T. HOWARD, F.R.C.V.S., London.

IN this age of veterinary medical associations, periodicals, new works, etc., it is a difficult task to find a subject of a comparatively unhackneyed character. The busy practitioner has, owing to the need for preparation, little time or opportunity for the performance of experimental or research work, on which to base a scientific communication. Original views on well-thrashed-out points demand for their production an ability almost akin to genius. However, the material utilised in this paper is original, at least with regard to its source.

The inspection has been conducted in the combined interests of the shipowners and the underwriters of the horse insurance, with a view to taking any steps, or submitting of any recommendations, that

would conduce to the well-being of the horses while on board ship and for a short period after their debarkation, and thus reduce the mortality and sickness to a minimum.

The method of procedure is to make a tour of the ship on arrival, and note the more serious cases; after which a more detailed inspection is made of each horse when walking on to the quay from the ship's gangway, healthy horses being allowed to pass on, while sick ones are set aside for diagnosis to be made at leisure.

The notes and observations that follow are the outcome of an experience extending over a period of four years, during which time over 45,000 horses have been inspected by the writer at the time of debarkation, about 1000 cases treated, and about 500 *post-mortem* examinations conducted on horses dying shortly after arrival in this country.

Conditions of the voyage and effects on horses.—The weather is undoubtedly an important factor. Otherwise healthy horses very rarely arrive with "filled" legs after a rough passage, the movement of the ship causing the horses to move, and circulation being thus assisted; the opposite is observed after a calm trip.

The condition of the atmosphere on the deck where the horses are carried is obviously a most important consideration. Not only have impurities resulting from respiration to be got rid of, but also gases, such as ammonia, etc., arising from the excreta, which are not removed until the ship arrives in port. Ventilating shafts (passing from the horse deck and opening on the non-covered-in part of the ship) and port-holes are provided with this intent, and serve the purpose well while the ship is in motion. In the event of the vessel coming to anchor through fog or any other cause, the ventilation becomes very defective unless large electric fans have been provided, which is very seldom the case. The atmosphere then becomes heated and malodorous; its effect on the animals is speedily made manifest by refusal of food, suffusion of the eyelids, and drooping heads; and those that have previously shown any tendency to "blow" will develop "pneumonia," or a kindred affection, from which they will speedily succumb unless relief is provided.

The state in which a cargo of horses lands is greatly dependent upon:—

(a). The carefulness with which infectious cases have been weeded out by the veterinary inspectors at the time of shipment in America; and

(b). Whether the animals were in a "seasoned" or "green" condition at that time.

It is an undoubted fact that there is much less mortality amongst the former than the latter, although at first sight one would imagine that horses at hard work on a highly nitrogenous diet, such as thoroughbreds and harness horses, would suffer more from a ten days' idleness than "green" bus or van horses. It might be urged that this is explained by the difference in breed, but against that explanation is the statement that seasoned bus horses have landed in South Africa in very much better condition than raw remounts.

Casual examination of a comparatively healthy consignment made at the end of the voyage would most likely fail to discover any sign of the recent journey. A closer inspection would reveal an absence

of "tone," or latent vitality, which is manifested in a variety of ways. Among these may be mentioned :—

- (1). Rapidly shown fatigue on exertion.
- (2). Small weak pulse (very persistent).
- (3). Liability to rupture of internal organs, etc., if animals are subjected to sudden or violent exertion.
- (4). The frequent occurrence of complete luxation of the patella.

Ruptured stomach has been most frequently seen, and next in order ruptured diaphragm, ruptured spleen, ruptured right auricle of heart, and ruptured posterior aorta.

Not one case of ruptured intestine is recorded with the foregoing, but a case possibly predisposed to by the conditions under review was intussusception of the small intestine.

The possibility of *post-mortem* accidents has always been considered in deciding as to the cause of death in these cases.

Mares in foal, especially towards the ninth month of gestation, frequently (sometimes three in a single trip) bear premature foals, which are usually dead. Fully developed live foals are sometimes born on the ship, but they seldom survive; at least one died from omphalothrombophlebitis.

Abnormally high temperature (2 or 3 degrees F.) occurs in many otherwise apparently healthy horses at the time of arrival; and on the following day, that is, after they have been landed about 24 hours, it has returned to normal. The explanation of this may be that it is caused by bacteria that require the assistance of the insanitary conditions prevailing in order to maintain their foothold in the animal body. The high temperature probably furnishes the chief cause of the premature labour above-mentioned.

It is here proposed to allude more particularly to some of the diseases and injuries incidental to this traffic, not so much with the idea of describing each one in detail, but rather of emphasising features that have arrested attention.

Diseases.—As will be surmised from the foregoing remarks relating to the atmosphere breathed by horses on ships, diseases such as pneumonia, influenza (and its complications), angina, pleurisy, pericarditis, and purpura hæmorrhagica, are by far the most frequent, especially pneumonia. Strangles, of course, may, and often does, exist with any of the preceding.

A disease which does not put in quite so regular an appearance as the above-named is an eye disease, which is probably specific ophthalmia; when noticed it is usual to find several cases on the same ship. Abdominal affections, with the exception of gastritis, enteritis, and fatty degeneration of the liver, are rare. Only two cases of volvulus are recorded in my notes on this subject. This apparently bears out the view that it is brought about by work, or the conditions of work.

Hæmoglobinuria is seldom seen in these horses while at sea, or during the first and second week after landing; such, however, unfortunately, is not the case when they are put to work.

Laminitis appears to be regarded by some as quite a common disease amongst horses at sea, but such has not been my experience. Occasional cases (not more than 1 in 1000) certainly occur, and usually with a fatal termination.

Gangrenous stomatitis is sometimes seen ; the sides and ventral aspect of the tongue become denuded of mucous membrane in patches up to 2 in. in size. The condition renders treatment difficult, as nourishment cannot be taken. The character of these cases is not infectious. Very possibly the complaint is not primary, but rather a symptom of purpura hæmorrhagica, the attack being of a non-typical character in other respects, which is not at all uncommon.

A few slight cases have been noticed where the inside of the upper and lower lips exhibited papules and vesicles—a condition sometimes regarded as variola equina. The cases were isolated, and no additional ones occurred ; which was a different result to what was once noticed in an English stable, when by the time the first case was found several others were also affected, and it was thought that the groom in charge had received the virus in his arm, where a vesicular eruption resulted.

A vesicular eruption, which, after the epithelium is shed, leaves a circular patch about the size of a threepenny piece, not at all uncommonly affects the skin around the lips and nostrils. From the time that they are first observed until they disappear probably not more than a week elapses. They do not give rise to any inconvenience to the horse, but cases are seen where they are present on the pigmented integument at the floor of the nostril. My reason for thus rather fully dealing with what, on the face of it, appears to be an insignificant complaint, is because the uninitiated might jump to the conclusion that it was glanders. Beyond the occasional situation in the neighbourhood of the nostril, there is no similarity whatever between the two diseases, but, to make assurance doubly sure, one or two cases have been malleined, with negative results.

Injuries.—The injuries met with are not numerous, unless the ship rolls badly during the heavy weather, and the horses break loose on account of the fittings giving way, as, if I remember rightly, happened on the transport s.s. *Rapidan* soon after leaving Liverpool for South Africa. The best livestock-carrying ships will and do bring horses through the roughest weather without the slightest injury.

When the dimensions (beam) of a ship do not allow the stalls to possess sufficient length, the horses' tails, owing to the motion of the ship, become rubbed against the end of the stall. The friction first removes the hair, and, if it continues, may cause a raw wound. The tail may become paralysed.

An idea is in existence that mange is a common complaint amongst imported American horses, but such has not been my experience, though it is quite possible that the above rubbed condition of the tail has given rise to the suspicion. Inflammatory exudation is produced by the rubbing, and forms a crust or scab which might very easily be mistaken for the crust present in an attack of psoroptic mange, of which the root of the tail is a common seat.

Gathered nail is a not uncommon injury. The accident occurs at the time of embarkation, and, if not noticed then, is very probably unnoticed until the animal is landed. Inspection will reveal a discharge of pus from the coronet, and on probing the hoof is found to be under-run, possibly everywhere, and ready to drop off. All degrees of this are seen.

These foot cases, even when severe, do well under treatment as

soon as the animal commences to recover from the effects of the voyage. This is possibly to be attributed to the fact that the animal, being confined to a limited space, is unable to move, and thus add to the damage already present. The carelessness with which carpenters drop their nails about when building the stalls explains this accident, but this will soon be a thing of the past, as new stalls which are permanent fittings of the ship are now being adopted, and these avoid the use of nails entirely.

An important aspect of the subject can now be dealt with, viz., the relationship between the equine health of this country and the importation of American horses.

Are American horses conveying any kinds of infectious disease not previously existing in this country? Or are they adding to the prevalence of those that are practically enzootic, and thus causing loss to the horse-owning community? Further, if such a condition of affairs exist, what is the remedy?

The answer to the first of these questions at the present time would probably be in the negative. An allusion to the introduction of "pink-eye" in the past is contained in an editorial in the *Veterinary Record*, 6th July 1901. It says: "The now well-known term of 'pink-eye' is American, and was introduced into Britain with an outbreak of a special form of influenza about the end of the seventies." Another paragraph in the same article says: "The horses imported across the Atlantic introduced two forms of ringworm which have caused endless trouble to dealers and job masters."

Cases of ringworm have been very rare amongst the horses in this inspection. Consideration of the fact that the horses are collected from over a large continent will make it apparent that the introduction of new types of disease must always rank as a possible contingency, and be provided against accordingly.

Parasitic Diseases.—Those parasitic diseases common to both imported and native horses may roughly be divided for the purpose of consideration into (a) those of bacterial, and (b) those of animal nature.

Several bacterial diseases have already been referred to, and most of them, with perhaps the exception of purpura hæmorrhagica, are infectious in nature, and are classed under the heading on that account, even although in individual cases the exact bacterium is still at large and unlabelled.

One can scarcely imagine any conditions more favourable to the spread of infectious disease than those which of necessity prevail on the average vessel.

As already mentioned, there is the unfavourable atmosphere. In many cases it is impossible to move a sick horse for isolation purposes. Horses are on either side of a sick horse, and others are facing within a distance of 3 feet. Most of these complaints are characterised by nasal discharge; consequently every time the animal coughs or attempts to clear the nostrils a fine spray of infection is played upon the animals in the immediate vicinity.

A point of interest perhaps not directly bearing on the point under treatment is the confirmation, by my own personal observation, of a statement made in the *Veterinary Record* some time ago, viz., that the croupous laryngitis associated with a clear straw-coloured discharge is

infectious to human beings. The attack lasts a few days, and is more uncomfortable than serious.

The variation in infective power and in severity of attack noticed in these diseases, especially in pneumonia and influenza, causes one to speculate as to whether each complaint has its specific organism or whether their name is legion.

A commonly observed occurrence may be given as an illustration. A ship arrives with a cargo of horses, of which possibly four are suffering from acute pneumonia; after leaving the ship they improve and ultimately recover. The next time that the same ship arrives it will be found that there are just about the same number of cases of pneumonia, but a great difference is observed as to the termination of the attacks; probably the lungs become gangrenous, and the cases all terminate fatally.

It may be that this is merely an example of that variation in infectivity and malignancy which is seen from time to time in so many other diseases—influenza of human beings, for example. English horses when infected from those under consideration usually take the various complaints in a much more serious degree; dealers in both British and American horses are aware of this circumstance, and take measures accordingly. It seems possible that a native horse may serve as a suitable soil for increasing the virulence of the disease received from an American horse, in a similar way to that observed when a new cow is introduced into a herd recovering from an outbreak of abortion.

Glanders.—If this contagious disease were common amongst imported American horses, it would either be necessary for the traffic to cease, or else it would be necessary to enforce strict quarantine regulations on arrival in this country (probably accompanied by the mallein test). The extra expense thus entailed would naturally raise the price to the purchasers, possibly beyond their capacity to buy; but this consideration could have very little weight if the equine health in this country were threatened.

Fortunately for American horse-breeders, etc., it is most exceptional for a glandered horse to be shipped to this country. The number of recorded cases of glanders in imported American horses is stated to be six (*Veterinary Record*, 6th July 1901). Some of these cases were traced to the breaking up of a large stud of working horses in an American town.

The majority of horses that cross the water are about five years old and have never been in town stables, which no doubt explains their comparative freedom from glanders. My assertion that they are so is based on the fact that among the 500 *post-mortem* examinations before mentioned, I have not found more than one pair of lungs that one might suspect of glanders, although the tubercles are always searched for.

Whilst glanders remains as prevalent in London as at the present time, the infinitesimal amount amongst the "Yankees" can be disregarded. The placing of restrictions on them would savour too much of "straining at the gnat and swallowing the camel," but a time will and must come when glanders will be stamped out of this country, and the meshes of the net will then have to be made so small that even an occasional case of glanders will be unable to enter the country.

The only way of doing that would be to mallein every horse after it has been allowed to settle down at a quarantine stable in the port.

The animal parasites infesting the alimentary tract of imported American horses do not include any varieties that are not observed in this country.

The gastrophilus is very constantly present on the cuticular portion of the mucous membrane of the stomach, and very often at the pyloric end of the duodenum as well; their number is often large.

The sclerostoma armatum and tetracanthum are comparatively common. *Post-mortem* examination where pneumonia has been the cause of death often reveals an accompanying enteritis due to the above parasites. Probably the debility caused by them rendered the animal more easy of conquest by the bacteria than would otherwise have been the case.

Such worms as the ascaris megaloccephala and the curved oxyuris are not nearly so frequently seen as in British reared animals.

The filaria papillosa is more common in American horses; the peritoneal cavity is the seat. The parasite is not very interesting from a practical standpoint, as it is never sufficiently numerous to give rise to symptoms.

The measures necessary for the prevention of the spread of the before-mentioned infectious diseases should consist of:—

(1). Strict veterinary inspection of all horses when landed, in order that all cases of sickness may be isolated and retained at stables in the docks for treatment until recovery or death occurs. The healthy animals might proceed to their destinations.

(2). A more rigorous veterinary inspection at the time of embarkation, in order that only perfectly healthy animals may be shipped.

(3). Examinations of mangers and fittings by veterinary inspectors, to insure that thorough cleaning and disinfection have been carried out.

(4). Ventilation on the vessels, in addition to the usual provision, to be regulated by electric fans in all parts of the ship where necessary.

(5). Railway boxes and trucks, both here and in America, to be officially inspected to insure thorough cleanliness and disinfection.

Some of the above measures are now in force, but there is still something to be done, and there is a prospect that when that has been accomplished the percentage of deaths, although now low, may vanish completely, and thus add one more laurel to the crown of the veterinary art.

SHIP-PNEUMONIA.

By E. E. MARTIN, Army Veterinary Department, Remount
Depôt, Calcutta.

OF the diseases that affect the horse on a long voyage, I think it will be acknowledged that pneumonia is by far the most serious ; in fact, I would estimate that more than 50 per cent. of losses on board ship result from this cause. I have found that it differs considerably in its characteristics from the disease as it occurs on land ; and it is with the view of obtaining the experiences of those who have lately done so much travelling with horse transports, and in that way clearing up the pathology of the disease, that I pen these notes.

There appear to be two forms of the disease distinguishable on board ship, the first characterised by its sudden onset and rapid course, and the second simulating more nearly the disease as it occurs on land. In the first variety the horse feeds well and behaves as if nothing were the matter until within a few hours of death. The history of such cases is that the horse cleared up the night's feed, and is found dead in the morning, or he is suddenly noticed very dull, with a high temperature and distressed breathing, and dies in a few hours. *Post-mortem* examinations in these cases show the lungs intensely congested, or, to use the common expression, "as black as your hat," and stinking.

In the second form it takes a much longer course, which varies from two or three days up to a fortnight. Besides the ordinary symptoms of dulness, raised temperature, and distressed breathing (sometimes none of these symptoms is well marked), a stench from the lungs on expiration is noticed, and there is usually a discharge from the nostrils. This discharge varies considerably in colour and quantity ; usually it is dark-coloured and gluey. The stench is a very constant symptom, and it is frequently the first thing that is noticed, for one of the peculiarities of the disease on board ship is that the animal often feeds well up to within a few hours of death. *Post-mortem* examination of this variety shows large green gangrenous patches throughout the lungs, with a considerable amount of pleuritic effusion in the chest cavity. Incision through these patches shows them to be full of solid and liquid debris of a dirty greenish colour, and with a very bad smell. The extent of these patches varies considerably, and I have frequently noticed that the rapidity of the course of the disease bears an inverse ratio to the area involved. Thus, a case that was landed a few weeks ago, and reported as having been ill for over a week, lingered on after landing six days ; on *post-mortem* I found the lower border only of each lung involved, the rest, with the exception of slight congestion, being perfectly healthy. As would be imagined, the areas most frequently involved by this gangrenous inflammation are the lower borders and anterior lobes of the lungs.

Some ten or eleven years ago I had to deal with an extensive outbreak of contagious pneumonia at Brighton, and again, a year or two after, among some mounted infantry cobs at Aldershot ; in both these outbreaks I was struck with the specific course the disease ran. It was the only disease I ever met with among animals that was

marked by a definite crisis. Its course ran from six to eight days, during which the temperature usually remained at 105° or over; and if the case lasted over the eighth day without a marked fall of temperature and generally improved symptoms, I knew it was going to die. In the cases that died it was very exceptional to have gangrene, and in all cases loss of appetite, except for occasional delicacies, such as carrots or green food, was a prominent symptom.

The main differences of the diseases may be shown in tabular form:—

<i>Contagious Pneumonia.</i>	<i>Ship Pneumonia.</i>
Specific course marked by a definite crisis.	Irregular course, occasionally taking a form that may be likened to a septic intoxication.
Appetite <i>nil</i> and constitutional symptoms well marked.	Appetite frequently remains good; constitutional symptoms irregular; generally stench on expiration.
Gangrene very exceptional.	Gangrene invariable.
A good percentage of recoveries.	Recovery extremely seldom.

In seeking the causes of this disease one naturally turns to the question of ventilation as the chief predisposing cause, and for several years I thought that bad ventilation and foul air were accountable for most of the cases of ship pneumonia; but on seeking evidence on this point I have had to change my mind, for as a general rule quite as many, if not more, cases occur among the deck horses as in those below. As the horses on deck are practically in the open air, one cannot accuse bad ventilation of invariably causing the disease. Occasionally in a badly-ventilated ship there will be a heavy mortality from this disease in the horses below which can be directly attributed to bad air; but these conditions do not obtain in most ships, for, as I mentioned before, I have actual figures to prove that as many cases occur on deck as below. The congestion that results in all organs from long standing in a cramped position is, I consider, the chief predisposing cause. This congestion seems especially to settle in the lungs and feet. Given the parts in a state of passive congestion, it is easy to understand what an excellent opportunity is afforded to germs (septic and otherwise) in an organ like the lung that is in direct communication with the air.

Other predisposing causes that affect principally the horses on deck may be mentioned, such as exposure to cold and wet. We know that it is extremely difficult to induce pneumonia or kindred diseases by exposure to the elements when the animal is in a more or less natural state, as, for example, when turned out to grass, but it is a different matter when cooped up on board ship; and as most voyages he is called upon to take, such as from North America to South Africa, England to South Africa, or Australia to India, involve a change of seasons, it is not difficult to understand that exposure may become a frequent cause of pneumonia.

Poorness of condition and debilitating diseases, such as strangles, catarrh, etc., must not be forgotten as predisposing causes.

The question now arises as to what part contagion plays in ship pneumonia. I have collected evidence on this point very carefully,

and have not been able to gather a single instance of anything that might approach being called direct contagion.

Almost all ships that have any large numbers of horses on board for any length of time seem to have a loss of from $\frac{1}{2}$ to 3 per cent. from this disease, and sometimes special circumstances, such as badly ventilated quarters, will cause a much greater percentage, but as a general rule it will be found that those cases that have occurred have come promiscuously from all parts of the ship. The point is one about which I should like to hear the evidence of others, but at present I do not think that segregation and disinfectant measures are likely to do any special good when a case occurs.

Is the exciting cause of the disease the same germ which is associated with contagious pneumonia on land? Not being a bacteriologist, and only having studied the disease clinically, I hesitate to give any definite answer to this question. I can only say I think not. From its clinical course I believe the cause must be some of the germs of gangrenous suppuration—anærobic germs, probably—which are pretty widely distributed, and may under ordinary circumstances be non-pathogenic. In this connection I would like to quote a few lines from a paper on "The Bacteriological Investigation of Fœtid and Gangrenous Suppuration," by Edward Rist, M.D., Paris, read before the British Medical Association and copied in the *Veterinary Record* of 26th October 1901:—

"Dr Guillemot has made a special study of pulmonary gangrene in general with the same methods, and has arrived at the conclusion that it is always caused by anærobic germs, and that the process of gangrene in general is a real process of putrefaction of the living tissues."

I look upon ship pneumonia as a true gangrene of the lungs. In the first form of the disease death appears to be the result of a septic intoxication due to the absorption of the products of putrefaction; in the second form it appears to be more of the nature of a septic infection. The exciting cause in both cases is the entrance of germs of putrefaction into a tissue in a low state of vitality owing to congestion.

I think, on the whole, that the term pulmonary gangrene would be rather more suitable than pneumonia.

As regards prognosis, I have found death the rule and recovery the exception. Occasionally cases that I have diagnosed as pneumonia on disembarkation have recovered after a prolonged convalescence, and I know no reason why small portions of gangrene should not become encysted and recovery take place, but I do not remember ever having seen a case that I could "smell" recover.

The preventive measures to be aimed at are everything that tends to prevent congestion of the blood. Foremost of these are exercise and grooming. Both these methods can be employed in carrying well-trained horses with plenty of attendants. I may say the only horse transports I have known land their horses at their destination without any loss from this disease are some transports bringing troops from China, and in these cases the horses were groomed and exercised daily. Where a large number of partially broken horses are taken with only a few attendants, exercise and grooming become much more difficult.

THE RELATIONSHIPS OF THE TUBERCLE BACILLUS TO THE OTHER ACID-FAST BACTERIA AND TO THE ACTINOMYCES.¹

By Dr MOELLER, Belzig.

THE discovery during the last ten years of acid-fast micro-organisms resembling the tubercle bacillus has made it impossible to identify the latter organism by simple microscopic examination, whereas previously it was customary to pronounce every rod-like organism that resisted decoloration with acids and alcohol as the tubercle bacillus.

It is true that the staining reactions of the leprosy bacillus had indicated that the tinctorial behaviour of the tubercle bacillus was independent of its other biological properties, and that in the course of time there might be discovered other bacteria resembling the tubercle bacillus with respect to the action of acids and alcohol.

There has been much discussion regarding the nature of this staining reaction and its connection with the special characters of the bacilli. Koch himself says that the special staining reaction of the tubercle bacillus is always valuable from a diagnostic point of view. It is, however, a great mistake to suppose that the etiological importance of the tubercle bacillus stands or falls with its specific staining reactions.

It has lately been shown by Klein, and more recently by Marmorek, that very young tubercle bacilli do not retain the colour in face of acids and alcohol. Marmorek ascribes this fact to the young tubercle bacilli being destitute of the envelope of fat and wax, which, on the one hand, makes it impossible for the ordinary basic aniline dyes to come into contact with the protoplasm of the tubercle bacillus, and, on the other hand, prevents acids and alcohol from abstracting the stain when once that has penetrated. Klein believes that the acid-fast property of the tubercle bacillus depends on the production of chemical substances by the body of the organism. These substances are absent from the young bacilli, which are therefore weak in the presence of acids. Berrel has succeeded in artificially depriving tubercle bacilli of their resistance to decoloration by acids and alcohol. Through prolonged exposure to warm xylol a wax-like material is extracted from tubercle bacilli, which are thereby deprived of their acid-fast character, although they retain their pathogenic properties. Whatever be the explanation of this acid-fast property of the tubercle bacillus it may safely be assumed that the like tinctorial behaviour of the other acid-fast bacteria admits of the same or a similar explanation.

Even this common staining reaction forms only a superficial bond; greater importance attaches to the fact that in respect of morphology and pathogenicity these bacteria more or less closely resemble one another and the tubercle bacillus.

The bacteria resembling the tubercle bacillus which have been known to us for a number of years are the leprosy bacillus, the smegma bacillus and the bacillus of an acid tubercle test.

¹ A paper contributed to the Proceedings of the Ninth Congress on Tuberculosis, 1901.

The leprosy bacillus was first described by Armanen Hansen in the year 1877. It was discovered in the leprosy nodules. It resembles the tubercle bacillus, but the rods are somewhat shorter. It is rather more easily stained than the tubercle bacillus; for example, it stains with watery solution of fuchsin at summer temperature, which is not the case with the tubercle bacillus. Veisser, who has specially increased our knowledge regarding the leprosy bacillus, gives Weigert's nuclear stain as the differential one. Neither Hansen nor Veisser succeeded in obtaining pure cultures. In 1887 Bordoni-Uffreduzzi claimed to have cultivated from the bone marrow of a leprosy patient a micro-organism which resembled the tubercle bacillus in its resistance to acids and in other respects. The cultures could not be carried on. The bacteria cultivated somewhat later by Babes, and recently by Czaplewski, from the organs of leprosy subjects differ from the true leprosy parasite in not being resistant to acids. Both these authors allege that the bacteria cultivated by them resembled the diphtheria bacillus. However, it is doubtful whether these bacteria cultivated from cases of leprosy are the true causal parasites of that disease.

The smegma bacillus was first discovered by Tavel and Alvarez, who in 1885 found it in the normal preputial smegma and also in the secretion of the outer integument, especially in those situations where an accumulation of epithelium can occur, such as the region of the anus and vulva, the groin, between the toes, etc. The discovery of the bacillus was made in the course of investigations made with the object of confirming the observations of Lustgarten in 1887 regarding the bacillus to be found in syphilis. It was found that the smegma bacillus morphologically and tinctorially closely resembled the syphilis bacillus described by Lustgarten, and the importance of Lustgarten's discovery was thus placed in doubt. Moreover, searching examination failed to show that Lustgarten's bacillus was constantly present in syphilitic tissue.

Tavel and Alvarez described the smegma bacillus as morphologically very similar to the tubercle bacillus, and stainable by the same methods as the latter, but less resistant to alcohol. Inoculation experiments on animals failed, as did also the attempts of the authors to obtain pure cultures. Lacer and Czaplewski, the former from the secretion of syphilitic affections, and the latter from gonorrhœal pus, have cultivated micro-organisms resembling the diphtheria bacillus, and declared by both authors to be identical with the smegma bacillus. This conclusion has recently been controverted by C. Fränkel. Fränkel believes that only the micro-organism which first attracted attention on account of its great resemblance to the tubercle bacillus should be regarded as the true smegma bacillus. This resemblance is lacking in the bacillus cultivated and described by Czaplewski and Lacer, and subsequently by Fränkel himself. On account of its form and other characters it ought rather to be designated a pseudo-diphtheria bacillus. From my own observations I can confirm Fränkel's views on this matter.

I have not been able to obtain a pathogenic effect in guinea-pigs with either the diphtheroid bacillus cultivated in a state of purity from smegma or the true smegma bacillus abundantly present in cutaneous secretion.

In respect of differential diagnosis, especially in relation to urogenital tuberculosis, the smegma bacillus has not great importance.

As another acid-fast micro-organism that has been known for a long time, I might here cite the bacillus of avian tuberculosis. It is distinguished from the bacillus of mammalian tuberculosis by the appearance of its cultures, and by its requirements in the matter of temperature. Whereas the bacillus of mammalian tuberculosis ceases to grow at 42° C., that of avian tuberculosis grows well at this and even a higher temperature. Fischel, who worked under Hueppe's guidance, declared both micro-organisms to be modifications (determined by conditions of nourishment) of one and the same species. Through the different physiological nutrient media—culture in the mammalian body in the one case, and in the warmer body of the fowl in the other—a departure from the normal is induced in both varieties. Fischel succeeded by artificial cultivation in bringing about an approximation of the two organisms in respect of external characters; he was able to accustom the bacillus of mammalian tuberculosis to a higher temperature, and on certain nutrient media to produce a similarity of appearance in the cultures. In respect of pathogenic properties, however, he was not able to convert the one into the other. Fischel reported that he was able to set up a general tuberculosis in guinea-pigs with the bacilli of avian tuberculosis, but the cultures obtained from guinea-pigs were identical with those of the avian bacillus. In similar experiments on animals I obtained the same results.

In the same sense, tuberculosis of cold-blooded animals is to be regarded as a modification of mammalian tuberculosis. By passing the bacilli of mammalian and avian tuberculosis through frogs Bataillon and Terre succeeded in inducing the organisms to grow at summer temperature. By the same method Lubarsch was able to modify mammalian bacilli so that for the cultures obtained from a frog's spleen the optimum temperature was 28-30° C. From fishes inoculated with the bacilli of mammalian tuberculosis Dubard also obtained cultures which grew at summer temperature. From the spleen of a slow-worm inoculated with sputum containing tubercle bacilli I obtained cultures which grew well at 20° C., but ceased to grow at temperatures over 30° C. In appearance the cultures resembled those of avian tuberculosis. I have not yet succeeded in re-acustoming them to a temperature of 37° C. by passage through warm-blooded animals.

A series of organisms in many respects resembling the tubercle bacillus have recently been recognised in the outer world.

The frightful frequency with which tubercle bacilli have been detected in butter and milk suggested that it would be well to ascertain whether the organisms found in these materials were always true tubercle bacilli.

Such investigations were first carried out by Petri and Rabinowitsch. Both of these succeeded in isolating from butter a bacillus which was resistant to acids and alcohol, but which was not the tubercle bacillus. The micro-organisms described by both these authors differed so slightly that it has been customary to speak of them in common as the "Petri-Rabinowitsch butter bacillus."

This bacillus takes the form of non-motile rods which are morphologically very similar to the tubercle bacillus, but sometimes rather thicker than that. Sometimes, as in the case of the tubercle bacillus, intensely staining granules can be recognised in the interior of the bacilli. In respect of staining reactions in coverglass preparations the butter bacillus behaves with the ordinary methods of staining, exactly like the tubercle bacillus. In sections it is not quite so acid-fast as the latter. The bacillus grows at summer temperatures. When incubated at the body temperature there is already a distinct growth after twenty-four hours. On ordinary solid nutrient media a pure culture of the butter bacillus is distinctly different from that of the tubercle bacillus, but in bouillon and on Proskauer's non-albuminous medium the cultures of the two organisms are very similar. When the butter bacilli are injected along with butter into guinea-pigs there is sometimes set up in the bodies of the animals lesions which both macroscopically and microscopically are easily mistaken for true tuberculosis. In most cases there is a peritonitis with extensive formation of cicatricial tissue. As a rule, the injection of pure cultures of the butter bacillus sets up slighter lesions in guinea-pigs than when butter is injected along with them.

According to Rabinowitsch, giant cells, nests of epithelioid cells, and typical caseation are never recognisable in the lesions. Animals infected with the butter bacillus never react to tuberculin.

Another acid-fast bacillus was isolated from butter by Korn. According to the description of that author, this bacillus differs from the Petri-Rabinowitsch butter bacillus morphologically, culturally, and especially in its effect on animals, and is therefore not to be identified with the latter organism.

Furthermore, within recent times acid-fast bacteria which do not differ materially from one another have frequently been isolated from butter and milk.

I have recently cultivated in a state of purity from milk a micro-organism which resembles the tubercle bacillus, and at the same time is essentially different from the grass bacillus II.

All the acid-fast bacteria which have been cultivated from milk and its derivations show a close resemblance to the grass bacilli. When one remembers the source of the latter, that is to say, their occurrence in the food of cattle, it seems justifiable to conclude that the milk and butter bacilli are varieties of the grass bacilli. The slight distinctions which they offer may be attributed to the effect of passage through the animal body.

I have repeatedly succeeded in cultivating these grass bacilli in a state of purity from the grasses commonly used for feeding cattle. Since they were first isolated by me from timothy grass (*Phleum pratense*) I named them timothy bacilli.

The timothy bacilli take the form of slender, sometimes slightly curved rods. It is not possible to distinguish them microscopically from the tubercle bacillus. Like the latter, they sometimes contain deeply stained granules. They show in their interior oval unstained places, they exhibit branching, and sometimes at one end club-like swellings. With the ordinary methods of staining, in coverglass preparations as well as in sections, the timothy bacillus stains exactly like the tubercle bacillus. It grows best at the body temperature, and

very badly at summer temperature. It thrives on all the ordinary nutritive media. When cultivated at the body temperature indications of colonies are already visible after thirty-six hours. In general the culture is distinctly different from that of the tubercle bacillus, but a resemblance is sometimes obtained when the timothy bacillus, after several passages through the animal body, is cultivated at a uniform temperature of 37° C. There is then observable an approximation to the slow growth of the tubercle bacillus. As regards pathogenic properties, the timothy bacillus sets up in guinea-pigs almost the same pathological alterations as the Petri-Rabinowitsch butter bacillus. The case is different with rabbits. When these animals are injected intravenously, or into an artery, with the timothy bacillus, lesions are induced which are very difficult to distinguish from a true tuberculosis. The formation of giant cells and epithelioid cells and the occurrence of caseation form a picture which is easily mistaken for true tuberculosis. Lubarsch says that beyond any doubt it is quite impossible to distinguish with certainty, either by histological or bacteriological examination, the tubercles caused by the timothy bacillus from true tubercles. It is only by the method of culture that the one can be distinguished from the other. None of the heifers infected with the timothy bacillus reacted to tuberculin.

One of the micro-organisms discovered by me on fodder grasses was named the grass bacillus II. It is essentially different from the tubercle bacillus, and the two organisms therefore cannot be considered identical. In respect of staining reactions the grass bacillus II behaves like the timothy bacillus or the tubercle bacillus. In old cultures it becomes not quite so resistant to acids. Morphologically it resembles the tubercle bacillus, but the rods are sometimes rather thicker. One of its peculiarities is a tendency to grow out into long threads, similar to those not infrequently met with in old cultures of the tubercle bacillus. In respect of the appearance of cultures the grass bacillus II closely resembles the Petri-Rabinowitsch butter bacillus. It thrives best at the body temperature, but it also grows at summer temperatures. In guinea-pigs it sets up lesions similar to those induced by the butter bacillus. It is most virulent when cultivated in milk. According to the investigations of Freymuth, a disease characterised by the formation of nodules is set up in cold-blooded animals (fishes, frogs, and lizards) by infection with the grass bacillus II. The bacilli found in the animal body have taken on a more slender form, so that they are morphologically not distinguishable from the tubercle bacillus.

I also reckon as a variety of the grass bacillus an acid-fast micro-organism found in animal excrement, and therefore termed the manure bacillus. I found it in dung which had been lying in the heap for months, as well as in the fresh excrement of cows, donkeys, and other herbivora. Morphologically and tinctorially the manure bacillus resembles the timothy bacillus. Culturally and in respect of its pathogenicity it more closely resembles the grass bacillus II.

To sum up, I would say that from a diagnostic point of view one may pass over the leprosy bacillus and the smegma bacillus on account of their relatively infrequent occurrence and the want of knowledge regarding their cultural characters. The organisms which are the

cause of tuberculosis in fowls and cold-blooded animals may be also passed over on account of their slight importance.

There therefore remain for consideration the grass bacilli, with their varieties, the milk and butter bacilli and the manure bacillus. Apart from slight differences all these resemble the tubercle bacillus in respect of their staining reactions, and the same may also be said regarding their morphology. Although the different varieties exhibit more or less variability of form, nevertheless it is often impossible to distinguish between them and the tubercle bacillus. The tubercle bacillus itself is also variable according to the medium on which it is grown. In innumerable examinations of sputum I have seen the tubercle bacillus sometimes shorter, sometimes longer, sometimes as thicker or thinner rods. C. Fränkel was the first to point out that in the florid form of tuberculosis the tubercle bacilli generally are present in the form of short rods. The less commonly encountered forms, such as threads, convoluted filaments, and club-shaped swellings, as well as unstained places, and intensively staining granules in the bodies of the bacilli, are all met with in the tubercle bacillus and in the other acid-fast bacteria.

Greater differences are exhibited in artificial cultures. Although now and again one may succeed in inducing a more or less close resemblance by the manner of cultivating, cultures of the tubercle bacillus in general exhibit such well-marked characteristics that they can without difficulty be distinguished from all others. Furthermore, there is a distinction in respect of the temperature necessary for growth, for, whereas all other acid-fast bacteria grow at summer temperatures, the tubercle bacillus requires a temperature approaching that of the body. The tubercle bacillus is also distinguished from all these others by its very slow growth. In the case of the other acid-fast bacteria distinct indications of colonies are already visible in twenty-four hours at the body temperature; but in the case of the tubercle bacillus, under the most favourable circumstances, such as with frequent reinoculation on specially selected media, it requires several days' inoculation to obtain a visible growth.

This exceptional behaviour of the tubercle bacillus is very valuable from the point of view of differential diagnosis. According to several authors, acid-fast bacilli, which investigation proved to be not true tubercle bacilli, have been found in the expectorate in morbid conditions of the respiratory organs, such as pulmonary gangrene. Fränkel and Pappenheim have found such bacilli at *post-mortem* examinations, and Rabinowitch showed by pure cultivations that the organisms probably represented a variety of the butter bacillus.

I have often found these pseudo-tubercle bacilli, not only in sputum from the lung, but also in nasal and pharyngeal mucus, sordes of the tongue and teeth, and plugs of material from the tonsils.

In such cases, where there are no physical symptoms, and the suspicion of tuberculosis rests solely on the presence of acid-fast bacilli in the discharges from the respiratory organs, a certain diagnosis may be made by the following simple procedure, which is based on the slow growth of the tubercle bacillus and the fact that a definite temperature is necessary for its cultivation.

The suspected material is mixed with nutrient bouillon, and kept for some time at about 30° C. If there is then observable a distinct

increase in the number of acid-fast bacteria it may be assumed with certainty that these are not true tubercle bacilli. Sometimes a multiplication of tubercle bacilli is observable when sputum is mixed with certain nutrient media and kept at the body temperature. This growth, which is perhaps attributable to the presence of globulin-like substances derived from the body, is always extremely slight, and it entirely ceases after forty-eight hours, whereas pseudo-tubercle bacilli exhibit a continuous growth at a temperature as low as 30° C.

No pathogenic effect was observed either with the bacteria isolated by Rabinowitsch from gangrene of the lung or with those found by me in the tonsil and in mucus from the throat and nose.

However, a differentiation of these pseudo-tubercle bacilli from those of true tubercle is often of more importance in affairs of practical life than in clinical cases. Of this I may give two examples.

For years a dread of using two of our most important articles of diet—milk and butter—was kept up owing to the view that all the acid-fast bacteria found therein were tubercle bacilli.

The recognition of acid-fast bacteria in the excrement of cattle was formerly regarded as conclusive proof that the animals in question were the subjects of tuberculosis, but this conclusion has been rendered untenable by the isolation of the manure bacillus.

With respect to pathogenicity, all the acid-fast bacteria have for a common property that they set up in the ordinary experimental animals a disease associated with the development of nodules, but while the pseudo-tubercle bacilli do this in only a limited number of cases and in particular circumstances, the true tubercle bacilli do it always. The general picture of a pseudo-tuberculosis of the animal body is macroscopically often so like a true tuberculosis as to be readily mistaken for that. There is, however, a distinct difference between the tubercles themselves. Whereas the true tubercles are of a firm proliferating character, the pseudo-tubercles have a more rapid growth and a tendency to the formation of abscesses. The typical histological characteristics of true tuberculosis—giant cells, nests of epithelioid cells, and caseation—are only rarely found in cases of pseudo-tuberculosis, viz., when the bacilli have been introduced into the animal body in special ways.

I may mention still another peculiarity of the tubercle bacillus. When one injects that organism together with butter into the peritoneal cavity of an animal the resulting lesions are not those of a typical tuberculosis, but take the form of a peritonitis with the production of much cicatricial tissue, quite similar to that which is set up by the injection of pseudo-tubercle bacilli in butter.

In spite of the many points of resemblance between the acid-fast bacteria and tubercle bacilli, the relationship is after all only a superficial one. In all that goes to make up the peculiar inner nature of the tubercle bacillus—its pathological effects on the human body and its etiological importance on tuberculosis—that organism occupies quite an isolated position among the acid-fast bacteria. Although pseudo-tubercle bacilli have on many occasions been detected in the human body, and even in diseased organs (pulmonary gangrene and affections of the tonsils), it has never appeared that they had in these positions any etiological importance.

Through artificial cultivation one can cause the tubercle bacillus and the timothy bacillus to approximate so closely in their outward characters that the one takes on features of the other. One can accustom the tubercle bacillus to a quicker growth at a lower temperature, and induce the timothy bacillus to exhibit the slow growth of the tubercle bacillus, so that their cultures take on a similar appearance, but in the end they remain two different species. One cannot convert the one into the other. However complete our technique may be, the best cultivator is still Nature, which cannot be overcome. What Nature has separated we shall hardly succeed in uniting.

A further peculiarity is exhibited by the tubercle bacillus in its method of propagation. It exhibits a saprophytic growth only when it is cultivated artificially. It is formed only in the products of disease or where these are present as a contamination, whereas the other acid-fast bacteria referred to in this paper have a saprophytic growth for their ordinary method of existence. The pleomorphism of the tubercle bacillus has been recognised for a long time. Soon after the method of recognising that organism was made known, several authors described instances in which the rods departed from the ordinary form although retaining the special staining reactions. Branched threads and club-shaped swellings were observed in cover-glass preparations from pure cultures, as well as from phthisical sputum and fragments from caverns in the lungs. More recently, through injection of animals, it has been possible to obtain in the tissues appearances which indicate a near botanical relationship of the tubercle bacillus with the radially arranged fungi. Babes described the occurrence of such form of the tubercle bacillus in rabbits inoculated subdurally, and Friedrich arrived at similar results by intra-arterial injection of the bacilli.

These views have been confirmed and extended by the later investigations of Lubarsch and Schultze, who have shown that such radiate arrangements of tubercle bacilli may also be found in various organs after local inoculation. According to the degree of virulence of the material used for inoculation, the manner of injection, and the earlier or later death of the experimental animals, more or less distinct streptothrix forms of the bacilli are observed. All authors recognise a great resemblance between these forms of the bacillus and the *actinomyces bovis*. According to Friedrich and Voske, a distinction is found in the fact that in the radiate colonies of the tubercle bacillus it is quite exceptional to find a direct connection between a bacillus and one of the clubs, whereas in the *actinomyces bovis* the recognition of a connection between the clubs and the mycelium is relatively easy.

The tinctorial behaviour of these streptothrix forms of the tubercle bacillus is variable, but the clubs are only quite exceptionally acid-fast. With the Ziehl-Neelsen method of staining it is not always possible to recognise the clubs, whereas good reliable pictures are obtained by the Gram-Weigert method and by the Birch-Hirschfeld stain for the *actinomyces*.

The exhaustive researches of Lubarsch have shown that the other acid-fast bacteria, of which the pure cultures always show threads, branched forms, and clubs in cover-glass preparations, produce

streptothrix forms when they are injected into animals, just like the tubercle bacillus. This affords proof of the close connection between the pseudo tubercle bacilli and the radiately-growing fungi, and therefore also of the relationship of the former to the tubercle bacillus. Although it has been possible artificially to induce the tubercle bacillus to exhibit the mode of growth that indicates its position among the more highly organised fungi, no one has yet succeeded in causing it to maintain this form. Perhaps in the course of time the conditions necessary for this may be discovered. In the meantime, the tubercle bacillus has the greatest importance when it occurs in that stage in which we know it best, viz., as a rod-shaped parasite.

EDITORIAL ARTICLES.

THE CAUSE OF TUBERCULOSIS, AND THE FALLACIES OF REASONING FROM ANALOGY.

IN last number of the *Journal* we published an article by Mr Goodall which dealt with the etiology and means of prevention of tuberculosis and the alleged transmissibility of the disease from the lower animals to man. We also published an editorial article in which reasons were given for dissenting from a number of the conclusions at which Mr Goodall had arrived. We now frankly admit that we wish we had allowed Mr Goodall's article to appear without criticism or comment, for we should then have been spared the necessity, as a concession to the author's aggrieved feelings, of publishing a rejoinder which, in point of rationality, falls below the level of the matter which is ordinarily admitted to these columns. In this rejoinder, which appears at an earlier part of this number, Mr Goodall suggests that the criticisms to which he objects can only be set down to a "temporary aberration" of the Editor's mind, with narrow-mindedness as a contributory cause. We are not going to offer any evidence to rebut the charge of mental aberration. That, of course, is only Mr Goodall's way of saying that we are entirely at variance with him concerning a number of questions raised in his paper, and when it is looked at in that way we see no reason why we should not plead guilty, with the reservation that the aberration is probably permanent.

On the other hand, we are not going to admit that we are incapable of taking the broad-minded view of the causation of disease which is habitual with biologists, including Mr Goodall. In passing, it may be observed that there is an amusing *naïveté* in Mr Goodall's attempt to defend his views regarding the cause of tuberculosis by classing him-

self with the biologists, who, he avers, "note cause and effect ever at work amongst the highest as well as the lowest forms of life," and to condemn those of the Editor by classing him with the bacteriologists, "who will not allow the possibility of any factors except bacteria exercising any influence on the causation of health and disease." In the first place, the Editor has surely as much claim as Mr Goodall to be regarded as a biologist, and, in the second place, the distinction drawn between biologists and bacteriologists in their manner of regarding the circumstances bearing on the cause of disease is wholly unjustifiable. No approach to the settlement of a controversy is ever made by suggesting that the opponent's views are those of a class who are habitually in error. With respect to the matters raised in Mr Goodall's first article, we did not say that the errors contained therein were those held by any particular class of persons; we took them as Mr Goodall's own peculiar views, and gave reasons for not accepting and acting upon them. It is true we did say that there was reason to fear that some of them were held by a good many other people, and indeed that was the main reason why we took the opportunity to correct them. That is also the reason why we propose to consider some of them again in the present article.

It will be observed that Mr Goodall defends the use of the compound adjective "popular scientific," which we were unable clearly to understand. What he says with regard to that point certainly calls for no reply. It makes painful reading, and, in Mr Goodall's interests, we could wish that he had not insisted on its publication.

The principal points raised in Mr Goodall's original paper, and rediscussed in the present one, were:—

1. The identity of the bacilli of human and bovine tuberculosis, and the transmissibility of the disease from the lower animals to man.
2. The relative importance of the tubercle bacillus and of other factors in the causation of tuberculosis.

With regard to the first of these points, the position taken up by Mr Goodall in his first article was that there were grave doubts as to the identity of the bacilli in man and animals, and that most probably the bacillus of bovine tuberculosis was not normally transferable to an animal of a different variety or family, or to man. In his second article he repeats this view in slightly different terms, his precise words being: "So far as at present known each animal has its own particular tubercle bacilli, not easily, by natural means, transmitted from animal to man." What was the evidence brought forward in support of these contentions? It comprised a number of alleged facts with regard to the non-transmissibility of parasites from one animal to another of a different species, most of them culled from the natural history of the animal parasites, but some from the domain of bacteriology. As regards the first of these, we did not

think it necessary to offer any criticism, considering it obviously absurd to infer that because the acarus of human itch cannot establish itself in the skin of the horse the bacillus of bovine tuberculosis is probably not pathogenic to man. With respect to the alleged specificity of most of the pathogenic bacteria—their restriction to animals of a single species—we accused the author of having overlooked or omitted to mention the highly pertinent fact that there were a number of exceptions to this rule—a number so considerable as to leave it perfectly possible that bovine tuberculosis was transmissible to man. We cited five different instances in which bacteria or other vegetable organisms were transmissible, and in natural circumstances transmitted, from animals to human beings, and pointed out the remarkable fact that every known bacterium that was common to two of the domesticated species¹ was also pathogenic to man.

These facts apparently have no importance in this connection for Mr Goodall. Indeed, his mind is so constituted as to lead him to regard them as further evidence that bovine tuberculosis is not transmissible to man. He is even thankful to us for having brought to his notice facts which he had overlooked, but which are so strongly corroborative of the position he had taken up! We do not wish to asperse Mr Goodall's intelligence, but, on the other hand, we owe something to our readers, and we feel that it would not be respectful to their mental powers to assume that any reasoned statement is necessary to obtain their assent to the proposition that glanders, anthrax, rabies, actinomycosis, and ringworm are examples that tell against the contention that bovine tubercle bacilli are not pathogenic for man.

We cannot allow Mr Goodall to pretend that he merely argued against the view that bovine tuberculosis is frequently transmitted to man. His contention was that in all probability it was "not transferable, normally," the obvious meaning being that it was not transferable in any circumstance arising naturally. He cannot have meant that bovine tubercle bacilli do not at present frequently find their way into the air-passages and alimentary canals of human beings, for if that had been his argument all his alleged analogies would have been doubly absurd.

It is necessary to call attention to this point, for in his remarks regarding rabies and the other diseases cited by us he mixes up two questions which had better be kept distinct. We are quite aware that he holds that the tubercle bacillus is harmless even to the ox, provided the animal is what he calls healthy and has a sound skin and mucous membranes, and we shall deal with that view presently; but he cannot have had this in his mind when he said that the bovine bacillus is not normally transferable to man, unless he is also prepared

¹ The word quadrupeds should have been used instead of species.

to maintain that the same bacillus is not normally transferable from ox to ox. It is therefore mere foolishness to attempt to dispose of the analogy furnished by rabies, by saying that human beings only contract that disease by inoculation, and that only a small percentage of bitten human beings develop hydrophobia. Although it has nothing to do with the point under consideration, we may notice the remarkable error into which Mr Goodall has fallen in assuming that every dog bitten by a rabid animal of its own species develops the disease, and that wounding of the skin is less necessary for infection in the dog than in the human subject.

We come now to the second point raised in Mr Goodall's two articles, viz., the relative importance of the tubercle bacillus and of other factors in the causation of tuberculosis. In the first of the articles it was held that tuberculosis was entirely a disease of weakly animals and of those predisposed by heredity, and, as a perfectly logical outcome of this view, it was maintained that tuberculin should not be regarded as the sovereign means of prevention, but that it was desirable to impress upon breeders the necessity of breeding and rearing young stock in conditions that would "make them proof against attack." Mr Goodall accuses us of having consciously and dishonestly represented his views on these points. There was no ground for such a charge in anything we said. It is possible, though it is not obvious, that we misunderstood Mr Goodall's meaning, but if we did the fault was more his than ours. He says that we wrongly ascribed to him the opinion that tuberculosis is attributable to some cause other than the bacilli, and that it is impossible to twist his words so as to convey a meaning so utterly foolish. What he did say was: "Let us go again to Nature's laboratory to show why tuberculosis is a cause of invasion, or the effect of a weakly constitution, for I believe it only attacks successfully the weakly, or what we are pleased to call those predisposed by heredity." The precise illustration which he then took was that of oak and elm trees whose roots had been interfered with by the making of banks and ditches, "so that the lowering of the vital energies is in these cases the cause of the invasion of the larvæ" (of the goat-moth). In criticism of these passages we said that it appeared to us to be nonsense to define tuberculosis as a cause of invasion of the tubercle bacillus, if the words had to be taken literally, and to that opinion we adhere. But we went on to say that what was probably meant was that tuberculosis is caused not by the tubercle bacillus but by a peculiar weak state of the animal body. We submit that in this we did no violence to Mr Goodall's words, and we are constrained to maintain, in spite of his denial, that he meant precisely what we suggested. He clearly meant that it was the interference with the roots rather than the larvæ of the goat-moth that determined the decay of the oak and elm trees, and the illustration was very

unfortunately chosen unless he also meant that the tubercle bacillus plays only a secondary rôle in the causation of tuberculosis. We repeat that this is a venerable error, and it is a pernicious error, since the question is not one of mere academic interest, but one of great practical importance.

As far as we can understand Mr Goodall, his position appears to be, (1) that there are two factors in the causation of tuberculosis, viz., the tubercle bacillus and a weak or unhealthy state of the animal body; and (2) that in the conditions of practical agriculture it is possible to breed and rear cattle so that they shall be proof against the attacks of the tubercle bacillus.

Now, it is obviously true that there are two factors in the causation of tuberculosis, viz., the tubercle bacillus and a state of the body in which only an ineffectual resistance is offered to the attacks of that organism. The same is true of every other bacterial disease, and even of such mechanical injuries as broken bones and sprained tendons. The kick of a horse may break a man's leg, but only if the leg is breakable. The quality of breakableness is thus a second factor in fracture of bones. In the same way the second factor in tuberculosis is the animal's inability to resist invasion by the tubercle bacillus. But everyone will admit that in the immense majority of cases of fracture in which violent mechanical injury is one of the factors, the other factor is of no importance. Why? Simply because bones unbreakable under the given circumstances are not to be found in the most perfect of animals. On the other hand, there are cases of fracture in which more importance is rightly attached to the quality of breakableness than to the mechanical force under which the bone gives way, as, for example, in cases of *fragilitas ossium*. In some of these cases the mechanical force under which the bone breaks is so slight that the fracture is termed spontaneous. Here the second factor acquires great importance, because the fragility is highly abnormal, that is to say, the immense majority of animals possess bones with far higher powers of resistance.

Now, Mr Goodall's view appears to be that the great majority of animals that fall a prey to the tubercle bacillus are in very much the same position as the animal suffering from *fragilitas ossium*, and he is therefore quite logical in regarding the tubercle bacillus as a factor of only secondary importance. Unfortunately, however, it appears to be only a "pious opinion" with him. *Fragilitas ossium* is rightly regarded as a cause of fracture, because there is the most unmistakable evidence that bones so affected become fractured in circumstances that have no injurious effect on the bones of ordinary individuals. But when we consider the incidence of tuberculosis in the same way, there is no evidence to prove that the second factor, or what may be called the degree of "vulnerability," has any important share in determining which individuals shall succumb to the attack and which

shall escape. At anyrate, we do not know of any, and none has been brought forward in either of Mr Goodall's articles.

In last number we pointed out that when a herd is tested with tuberculin, or when we make a *post-mortem* examination of cattle killed in a slaughter-house, we often find that individuals conforming to all our notions of health and normal conformation have contracted tuberculosis, while others of the same herd that are much below par in respect of general condition and conformation have escaped infection. This Mr Goodall admits, but he endeavours to extricate himself from the position in which the admission places him, by asserting that "from Nature's point of view these apparently healthy animals are weaklings and monstrosities." Apparently Mr Goodall does not see that in this he has been guilty of a very obvious controversial trick. The point under discussion was whether healthy normal individuals are susceptible to tuberculosis, and when he is confronted by evidence proving that they are, he demands that every tuberculous animal shall be considered a weakling and a monstrosity, "from Nature's point of view." He thus begs the question by so defining "healthy," that any search for evidence is bound to prove that only abnormal, unhealthy, or weak individuals contract tuberculosis. Mr Goodall must remember that he is not Nature, and what he should have said was that from his own point of view every tuberculous animal was a weakling and a monstrosity before its infection. But when put in that way the matter is not important. Perhaps he also believes that the only individuals not regarded as weaklings and monstrosities by Nature are those that effectually resist every adverse influence brought to bear on them, from railway accidents to deadly poisons.

It will be observed that Mr Goodall attaches very great importance to breeding from immature animals as a cause of tuberculosis, and he insists that the custom of breeding from such animals is almost universal. This, we are asked to believe, is another thing abhorrent to Nature's eye. Upon this point we have two comments to make. In the first place, assuming that the word Nature as here used does not mean Mr Goodall, we submit that there is no evidence that Nature has any abhorrence of reproduction by animals that have not reached maturity. On the contrary, there are many species which, in a state of nature, breed before they have attained their full development. An animal cannot breed until Nature has provided it with the sexual appetite and the necessary reproductive cells, and when these have been so provided it would be very inconsiderate on the part of Nature to abhor the exercise of the reproductive function. To be quite fair, we will admit that in a state of domestication a larger proportion of cattle are prematurely mated than would be the case among the same animals running wild, but that is very far from saying that premature breeding is a universal custom in this or any

other country. We maintain that in this country the calves born of immature parents form a very small minority.

The second comment which we have to make in this connection is, that there has not yet been produced a scrap of evidence to show that the progeny of immature parents are in any special degree predisposed to tuberculosis. No one can seriously maintain that there is any obvious reason why they should.

The spermatozoa produced by a yearling bull may not be so numerous as those produced by a four-year-old one, but they may be individually as good in the one case as in the other, and, after all, only one sperm cell is concerned in fertilisation of a single ovum. Mr Goodall thinks that it would be well to collect statistics showing the influence of premature breeding on predisposition to tuberculosis, but apparently he does not possess any himself. In the circumstances, it is impossible to imagine what are the grounds for his assertion that breeding from young animals is largely responsible for the present wide prevalence of tuberculosis among cattle.

Lastly, we have to discuss the possibility, in the conditions of practical agriculture, of breeding and rearing animals of the bovine species so that they shall be "proof against attack" by the tubercle bacillus. What evidence is there that such a thing is possible? We unhesitatingly answer, None. Mr Goodall has not produced any and presumably does not know of any. He tells us that in his own vigorous state of health he believes that tubercle bacilli could not make any impression on his body. But that is not trustworthy evidence as to his invulnerability by the tubercle bacillus, and we earnestly hope that he will not take any avoidable risks of infection. Are cows and bulls never to be mated under four years old, and animals of all ages to roam the fields and breathe fresh air all the year round? Farming under such conditions would be impossible, and even if such measures were practicable there is no reason to believe that they would lead to the production of a race of animals proof against attack by the tubercle bacillus. Among the prairie cattle of North and South America and Australia tuberculosis is comparatively rare, but there is abundance of evidence to prove that they have no immunity, and that their escape is mainly, if not entirely, due to the obstacles which an out-of-door existence places in the way of infection.

To sum up, we have nothing to take back from the criticisms which we made in last number on Mr Goodall's plan for exterminating tuberculosis. In his second article he has wrongly accused us of having counselled the neglect of general hygienic measures in dealing with tuberculosis. We have never written anything in these columns that will bear that interpretation. Dirt, darkness, over-crowding, and bad ventilation favour tuberculosis by smoothing the paths of infection, but the most perfect sanitary conditions possible in a cow-shed will

not eradicate tuberculosis from a herd, or prevent the spread of the infection when tuberculous and healthy animals are housed together. Hence the justification for the assertion that the man whose sense of proportion is not hopelessly defective will attach minor importance to everything except the operation of contagion.

THE A B C OF SWINE-FEVER.

FROM reports of speeches that have recently appeared in the daily Press, we gather that the Right Hon. R. W. Hanbury, M.P., President of the Board of Agriculture, has a poor opinion of the knowledge of the veterinary profession regarding swine-fever. He is reported to have said, at the half-yearly General Meeting of the Royal Agricultural Society of England, that he had been able to recognise that there was a great deal of ignorance of the nature of this disease not only in England, but also on the Continent. At the annual dinner of the Central and Associated Chambers of Agriculture this opinion was delivered in a phraseology that was rather more offensive, for on that occasion Mr Hanbury is reported to have said that he had been able to satisfy himself that the veterinary profession were ignorant of the A B C of swine-fever.

The President did not explain how he had been able to gauge the knowledge, or want of knowledge, of British veterinary surgeons on this important matter, and we cannot believe that his adverse opinion is founded on an inability of his own professional advisers to furnish him with the information which anyone possessing an elementary knowledge of the disease ought to be able to supply. He did, however, explain that he was able to bring Continental veterinary surgeons under his charge of ignorance because the recent Congress on Tuberculosis had presented him with the opportunity to discuss the subject of swine-fever with representatives of foreign veterinary science. Should the report of Mr Hanbury's speeches meet the eyes of these distinguished foreigners they will probably be surprised to learn the use to which their remarks on the subject of swine-fever have been put. It cannot be pleasing to them to know that while perhaps flattering themselves that they were unfolding their replete stores of knowledge regarding swine-fever and Continental methods of dealing with it, they were merely affording the President of the British Board of Agriculture an insight into their abysmal ignorance of the disease.

Ignorance is a harsh word, and it is unpalatable to be told that the veterinary profession does not yet know even the A B C of a disease that has been rampant in this country for half a century. It may confidently be stated that this is not the opinion which the veterinary profession entertained of its own knowledge on the subject of swine-

fever, but, of course, that does not disprove the charge of ignorance. The densest ignorance is generally accompanied by perfect contentment with the amount of knowledge possessed, and by an inability to conceive that there is anything further to learn. The first step towards improvement in such a case is to bring home to the person concerned the fact that he is ignorant, and harshness of language in the process is even excusable. Moralising in this way, one is led to ask whether Mr Hanbury, in the guise of a candid friend, has not after all done the veterinary profession a good service by pointing out to its members that they do not know even the A B C of swine-fever.

This raises the question, What is the A B C of this or any other disease? In this connection it must be remembered that swine-fever is an admittedly contagious disease, and that Mr Hanbury's recent deliverances regarding it were made *a propos* of the difficulties experienced by the Board of Agriculture in stamping it out. Now, in order to wage a successful war against a contagious disease, it is not necessary that our knowledge regarding it should be so complete that nothing remains to be discovered. As a matter of fact, only the imperfectly informed would venture to say of any disease of man or animals that the future cannot add anything to the knowledge of it now existing; and in this country we have succeeded in stamping out several diseases regarding which the veterinary profession would be the first to admit that much remains to be learned. Pleuro-pneumonia, rabies, cattle-plague, and foot-and-mouth disease are examples. Presumably Mr Hanbury would admit that the veterinary profession were not ignorant of the A B C of these diseases, and must therefore have meant that they are less well informed regarding swine-fever.

What facts may be said to have been definitely established concerning the pathology of swine-fever? In the first place, it is known to be a bacterial disease, and the organism which is the cause of it is known. At least in the veterinary profession it is also regarded as an ascertained fact that in this country the disease is a purely contagious one, and that outbreaks of it are never sporadic. The existing information relating to the period of incubation, the symptoms exhibited, the duration of the disease, and the lesions characteristic of it, is fairly complete—certainly as full as it is regarding any other contagious disease of the domesticated animals.

As already admitted, it would be absurd to maintain that in veterinary pathology swine-fever represents a closed chapter, but we are also bound to say that we cannot agree with Mr Hanbury that the A B C of the disease has still to be learned by the veterinary profession.

We must, therefore, reject the hypothesis that veterinary surgeons here and on the Continent are incapable of measuring their own ignorance regarding swine-fever. The fact appears to be that the lack of

knowledge—we hesitate to say ignorance—is on the part of the President of the Board of Agriculture, who has not yet found time to master the A B C of the disease. We do not doubt that, if he will take the trouble to consult the members of the profession whose special duty it is to advise the Board regarding veterinary matters, he will find that much more is known about swine-fever than he at present supposes. It is even possible that he might arrive at the conviction that the failure of the Board's operations against the disease is less due to the ignorance of the veterinary profession than to the obstinate pursuit of a policy that ignores fundamental facts with which most veterinary surgeons are well acquainted.

We are told that the Board is now engaged in carrying out a more scientific investigation concerning swine-fever than has ever been made before, and that it has also on hand an *exhaustive* investigation of glanders, and an enquiry into louping-ill and braxy which was launched with a promise that the cause of these diseases and a remedy for them would be discovered. These are dangerous terms in which to describe investigations that have scarcely been begun. We earnestly hope that the results of them will not be disappointing, but we cannot help thinking it is a mistake to trumpet that the mountain is in labour.

PROFESSOR KOCH'S PAST AND PRESENT OPINIONS REGARDING THE RELATIONSHIP BETWEEN HU- MAN AND BOVINE TUBERCULOSIS.

It will be remembered that at the recent British Congress on Tuberculosis Professor Koch startled the scientific world by declaring that "human tuberculosis differs from bovine, and cannot be transmitted to cattle." On the same occasion, as if to anticipate a charge of inconsistency, he said, "Even in my first circumstantial publication on the etiology of tuberculosis I expressed myself regarding the identity of human tuberculosis and bovine tuberculosis with reserve. Proved facts which would have enabled me sharply to distinguish these two forms of the disease were not then at my disposal; but sure proofs of their absolute identity were equally undiscoverable, and I therefore had to leave that question undecided."

Since the date of the Congress some too enthusiastic defenders of the view that the two diseases are distinct have brought a charge of misrepresentation against those who, in criticism of Professor Koch's present attitude, pointed out that Professor Koch himself was mainly responsible for the view generally held during recent years, namely, that human and bovine tuberculosis are identical diseases. Those who bring this charge appear to be under a double misapprehension. In the first place, no one has ever maintained that the two diseases

were generally regarded as identical because Professor Koch had stated that such was his own opinion. There are no Popes in science, and the mere *ipse dixit* of one man, however eminent he may be, is not sufficient to upset convictions arrived at after examination of the whole available evidence. What is meant when it is said that Professor Koch is mainly responsible for the generally accepted opinion that bovine and human tuberculosis are identical diseases is, that the published account of Professor Koch's own researches appeared to most people to compel that conclusion. The evidence lay in the new facts brought to light by Professor Koch's researches, and not in any degree in the conclusions which that distinguished authority drew from these facts.

In the second place, however, Professor Koch, in spite of his recent disclaimer, did assert, in terms which to most people appeared quite unambiguous, that in his opinion the two diseases were identical and intercommunicable. In proof of that it is only necessary to quote the following two paragraphs from Professor Koch's "first circumstantial publication on the etiology of tuberculosis," which appeared in the *Berliner Klinische Wochenschrift* for 1882 (p. 230):—

"Tuberculosis of the domesticated animals, and especially bovine tuberculosis, is undoubtedly another source of infection. This fact indicates the position which, in the future, hygiene must take in connection with the danger of the flesh and milk of tuberculous animals. Bovine tuberculosis is identical with human tuberculosis, and is thus a disease transmissible to man. It must therefore be treated like other infectious diseases transmissible from animals to human beings. Be the danger which arises from the consumption of the flesh or milk of tuberculous cattle ever so great or ever so small, it exists, and it must therefore be prevented. It is sufficiently well known that anthrax flesh is often consumed by many persons for a long time without any injurious result, and yet no one concludes therefrom that the traffic in such flesh ought to be permitted.

"With regard to the milk of tuberculous cows, it is worthy of remark that the extension of the tuberculous process to the mammary glands is not seldom observed by veterinary surgeons, and it is therefore quite possible that in such cases the tuberculous virus may be immediately mixed with the milk."

The above paragraphs may be commended to the notice of the correspondent who wrote as follows in a recent number of the *Veterinary Record*: "Professor M'Fadyean foolishly said at the Congress, 'I thought, Professor Koch, when you had found a bacillus as the cause of tuberculosis in man, and a bacillus in the cow, you came to the conclusion that they were one and the same.' 'No,' says Koch, 'I never indicated such a conclusion.'"

THE HISTOLOGY OF BRAND-CANCER.

IN an article by the Editor which appeared in a former number of this *Journal* (Vol. IV., p. 244), reference was made to the so-called brand-cancer—the tumour-like growth which occasionally forms on the skin

of cattle where they have been branded with the hot iron. The name has come into use among stock-men in Australia, and the fact would seem to indicate that the formation of such growths is not a rare occurrence. In the article referred to a description was given of the histology of one of these tumours, of which a small piece had been given to the Editor by the late Mr Archibald Park of Hobart Town, Tasmania. In that particular case the growth had a typically carcinomatous structure, and in view of the interest of this fact, from its bearing on the etiology of the carcinomata, a hope was expressed that an opportunity would be obtained to examine other tumours of the same kind. That opportunity has just been afforded to us by the kindness of Mr Desmond, Government Veterinarian and Chief Inspector of Cattle, Adelaide, S. Australia, who has forwarded stained and mounted sections from two different cases of brand-cancer.

The first case is described by Mr Desmond as follows :—

Brand scar from the skin of a fat bullock. Seat, upper portion of ribs; extent, 20 inches long by 7 inches wide; appearance, horny growth, 3 inches long. The animal was condemned as unfit for food, but a most careful examination failed to reveal any secondary growths.

The second case was one of brand scar from the rump of a fat bullock. The lesion was breaking down towards its centre, and the subcutaneous tissue under it was in a state of inflammation. No secondary tumours.

We have examined the sections from these two growths, and find (1) that they have a practically identical structure, and (2) that they have not the histology of carcinomata. On the contrary, the disposition of the epithelium, which is very abundantly present, is that which is found in papillomata, or in chronic inflammatory lesions of the skin with abnormally active proliferation of the cells of the rete mucosum. The papillæ of the cutis vera have evidently been much distorted as the result of some chronic irritation, and the surface on which the cutaneous epithelium rests is consequently much less regular than in the normal skin. Consequently, also, strings of epithelial cells are found at quite abnormal distances from the surface, but there is no evidence that these cells have broken away from the epidermis or are invading the connective tissue. The luxuriant epithelial growth at many places shows signs of degeneration, and in it there are numerous collections of leucocytes.

If a guess may be hazarded from the structure of these specimens, we would say that brand-cancer is probably, as a rule, an inflammatory lesion of the skin, the result of some chronic irritant implanted in the branded area before repair has been effected, and that the irritant is probably bacterial.

Reviews.

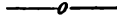
Bovine Obstetrics. By M. G. de Bruin, Instructor of Obstetrics at the State Veterinary School in Utrecht. Translated by W. E. A. Wyman, M.D.V., V.S. London: Ballière, Tindall & Cox, 1901.

IN translating into English Professor de Bruin's excellent text-book on Bovine Obstetrics, Dr Wyman has laid British and American veterinary surgeons and students under a debt of gratitude. The work represents the happy medium between the booklets which are adapted for cramming purposes by the student, and the ponderous tomes which, although useful to the teacher, are not exactly suited to the requirements of the everyday practitioner. It extends to 377 pages, and contains seventy-seven excellent illustrations. It treats in succession of the physiology of normal pregnancy and parturition, the diseases and accidents of pregnancy, the morbid conditions of the foetus and its annexes, abnormal parturition and obstetrical operations, the diseases apt to follow labour, and the special affections of the young calf. Both translator and publisher have done their work in a way that deserves praise, and we can strongly recommend the work to veterinary students and practitioners.

A Manual of Determinative Bacteriology. By Frederick D. Chester, Bacteriologist of the Delaware College Agricultural Experiment Station, and Director of the Laboratory of the State Board of Health of Delaware. New York: The Macmillan Company, 1901.

THE author of this work has set himself a very difficult task. In the preface he says: "To the student working in the laboratory the determination of unknown bacteria has been almost impossible, except with the expenditure of an amount of labour which was impracticable. With the use of the present manual it is believed that the teacher can place a given culture in the hands of his pupil and expect him to determine it, as is done with other organic forms." We doubt whether any experienced teacher of bacteriology will share this very sanguine expectation. The author has laboriously collated and condensed the description of almost all the known pathogenic and saprophytic bacteria, and for certain purposes the work will undoubtedly be useful; but when it is stated that the account given of many important organisms is contained in ten lines or less, the absurdity of expecting a student to identify a given culture by the aid of the information herein given, becomes apparent. For example, only six lines are given to the characters of Bang's abortion bacillus, and the most important of its cultural peculiarities, without which it cannot be identified, is omitted. Moreover, there are serious sins of commission as well as of omission, such as the statement that the bacillus of quarter-evil is stainable by Gram's method.

CLINICAL ARTICLES.

**A NOVEL METHOD OF RESTRICTING THE MOVEMENTS OF HORSES' LIMBS DURING TREATMENT FOR INJURIES.**

By CHARLES SHEATHER, F.R.C.V.S., London.

I HAVE designated this system of restraint "novel" because I think it will prove absolutely new to the members of the veterinary profession, excepting some who may have seen it in application in my own practice.

There can be but few who have not at some time experienced difficulties in the treatment of injuries to horses' limbs, for which

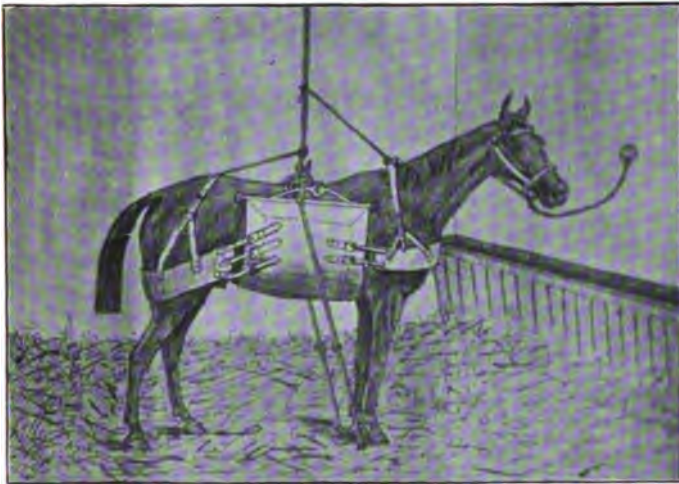


FIG. 1.

Showing the apparatus in use.

absolute rest or modified and restricted movement was most essential to recovery. It was in 1880 that I first schemed the plan delineated in the illustrations, which then, as in many cases since, proved of incalculable benefit.

In the spring of this year a roan mare, the property of the West End Cab Co., kicked in harness, perforated the dash board with her off hind leg, sustaining a long, deep, oblique wound across the front of the hock, traversing the bend. After six weeks' ordinary treatment the wound had made no real progress; a deep, ragged cleft,

free from discharge, but without any sign of filling up, remained. It was due simply to the fact that flexion and extension of the hock could not be prevented, and so the fissure persisted. I thought that, if I could only prevent the movement, healing by granulation might follow. I could see that if the heel could be prevented from moving from the floor, a great amount of rest must ensue. To this end, I placed the mare in slings, and had a bar shoe made to fit the foot, with the bar extended outwards for four inches beyond the hoof, and somewhat curved upwards from the ground (fig. 2, A); in this extension was punched a round hole about half an inch in diameter. A rod was made of three-eighths of an inch round iron, with a collar and slot at its lower end (fig. 2, B); the end of the rod would fit easily in the hole, and allow a split key (fig. 2, C) to be inserted in the slot to prevent its removal, while the collar prevented its passing too

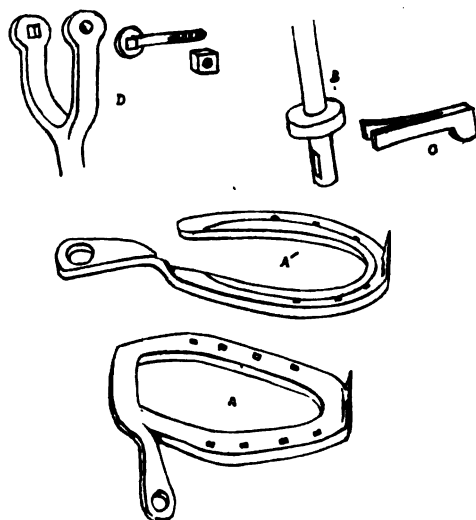


FIG. 2.

For description see text.

far through. - At the upper end of the rod, which was of such a length as to extend from the foot to the bar on the end of the girth of the sling, was shaped a V piece or jaw, traversed at its upper part by a bolt, for which suitable perforations were made in the jaw (fig. 2, D). This gripped the bow-like portion of the bar which, in all my slings, rises from the straight bar on the end of the girth, to receive the hook end of the main cross-bar, the hook being received in an eye placed two-fifths of the breadth of the girth from its anterior end.

The shoe was pulled off once because the rod from the shoe to the jaw was not long enough; and the rod was badly bent twice because it was made of three-eighths of an inch rod, which I corrected by making it of three-fourths of an inch rod.

The plan was successful, for under three weeks the wound had healed. The restraint was sufficient to prevent flexion of the hock,

but not sufficient to provoke that feeling of fixation which is so distasteful to animals; the mare could shuffle about on the floor, change her position, and give herself a certain amount of ease and satisfaction by movement, but she could not get her heel off the floor, nor could she flex the hock.

A long succession of cases, including all sorts of injuries, equally successfully treated, could be related, occurring since 1880; one more may probably suffice.

In 1894 a thoroughbred stallion, contrary to all expectation, and possibly to the disappointment of some, made a most successful recovery under this plan.

When first seen he had "broken knees" of three months' standing, deep horizontal fissures extending right across the knees, with the surrounding parts enormously swollen. His case was regarded as hopeless; he had got out of all sorts of slings, had been tied up till he fell, and had reached that stage of exasperation which only thoroughbreds can effectually exhibit. The treatment had degenerated into squirting an astringent lotion at his knees, from a distance of ten or twelve feet.

I realised at once that no treatment could be effectual unless movement were restricted; slinging and the application of the leg-irons were advised. The suggestion was received with ill-concealed ridicule. Had he not put slings at defiance, by bursting them and extricating himself from them? In the end he was sent to London, received a low diet in which was concealed considerable amounts of narcotics, was successfully placed in slings, and had the special shoes and leg-rods fitted, the shoes being of the form shown in fig. 2, A', which are simple to make, and very suitable for the fore feet.

As the effects of the narcotics passed off, he realised that he was under restraint, and started kicking; this was promptly checked by hauling him entirely off his feet by the sling pulley; as soon as he quieted down, he was lowered. He repeated the kicking at intervals for two or three hours, but alternate suspension and lowering made him think better of it. During all his violence not a single thing gave way. He became very tranquil, allowed his knees to be cleansed and dressed, and in seven weeks returned to his owner with wounds healed. In the treatment of this case I received the most able assistance from Mr C. E. Edmonds, who was at that time my senior assistant.

This method is not difficult to apply, it allows of a certain amount of shuffling about and shifting of position without flexion of joints, with one exception; the knee may be flexed somewhat by relaxing and dropping the shoulder, and the pastern may be drawn up into an upright position. Both these positions are easily defeated by passing a broad band round the leg above the knee, and attaching it to the eye worked in the rod for the purpose.

This particular form of fixation will suggest itself in those cases of fractured or injured ulna, laceration of shoulder muscles, and to correct the "dropped shoulder" position attendant upon anterior rib fracture. The main points in its application are:—the rod must be carefully adjusted as to length, it must drop into the hole in the shoe extension quite easily, and its diameter should be three-fourths of an inch. The shoe must be well nailed on.

When applying it to the fore limb, it must sometimes be curved outwards and backwards opposite the fetlock joint, to avoid contact with this joint in certain conformations. The jaw at the top prevents this curve getting the wrong way round.

This system will be found far more easy of application and retention than all the splints and bandages ever applied. It is unsuitable for any case suspected of fracture in the lower bones of the limbs.

CASE OF BOTRYOMYCOSIS IN A HORSE.

By JOHN B. WOLSTENHOLME, F.R.C.V.S., Manchester.

PATIENT, a red roan cart gelding ; ten years old. Part affected, the skin and subcutaneous connective-tissue at the lower posterior part of the left thigh.

For four years the horse had worked in the shafts of a four-wheeled heavy vehicle ; but at the latter part of 1900 he was put to work in chain trace gears.

The "spreader" or "stretcher" of the traces would ride or be carried on that part of the thigh which is affected ; this may have had some part in causing the lesion.

In November 1900 a small boil, about the size of a hazel nut, formed over the part, and then burst. From this time onward others formed and gave exit to small quantities of pus, which quickly dried.



It was 12th June 1901 when he came under my notice, at which time there were three or four points of suppuration, several cicatrices, and considerable thickening of the skin and subcutaneous tissue around.

At the end of October, when the accompanying photograph was obtained, the area of disease had spread until it occupied both the inner and outer aspects of the thigh, and extended in a perpendicular direction $11\frac{1}{2}$ inches, and $13\frac{1}{2}$ inches across.

The skin and subcutaneous connective-tissue is thickened and studded with eighteen depressions of about five-eighth inch diameter, which represent a corresponding number of abscesses and ulcers, most of which are healed.

When an abscess bursts a pale yellow viscid pus escapes, a portion of which dries on the wound and forms a firm crust.

Examination of the pus shows the presence of a number of grey opaque points, just visible to the naked eye, which when transferred to a slide and examined under a one-inch objective are seen to be colonies of the *botryomyces equi*.

They are easily examined in a drop of water or glycerine, but come out beautifully when stained with rubin and orange.

Of half-a-dozen colonies measured, the size varied from 0.2 mm., to 0.45 mm.

INTRACEREBRAL INJECTION OF ANTITETANIN FOR THE CURE OF TETANUS IN THE HORSE.

By SIDNEY VILLAR, F.R.C.V.S., Harrow.

CASE I.—On 9th June of the present year a small black male pony was brought to my Infirmary affected with tetanus. The animal was distressed somewhat by its walk of 3 miles, the disease was in its initial stage, and, although the usual symptoms were well marked, it quickly got over the effects of the journey.

The course of the disease in this case was subacute. The pony was put in a loose box, kept perfectly quiet, and had solution of extract of belladonna daily in its drinking water, being meanwhile well nursed and supplied with suitable food.

It continued to get gradually worse, and, as on the 17th June trismus was becoming very marked, I determined to resort to the intracerebral injection of pure antitetanin¹, the pony being a suitable subject for this operation on account of its small size and gentle disposition.

On the morning of 18th June, having well washed the skin of the forehead with perchloride of mercury soap, shaved it as closely as the movements of the pony would permit, and having injected subcutaneously 3 grains of cocaine in solution, I incised and dissected an angular flap of skin about one-and-a-half inches in length, and cut through the temporal muscles and for a small space detached them from the bone beneath, thus exposing a small portion of the right parietal bone at a distance of one inch from the parietal ridge, on a line with the anterior margin of the root of the ear. With an archimedian drill, bought at an ironmongers for 1s. 6d., a small circular hole was made through the bone, the length of the drill being previously limited to rather more than an inch by threading a cork on it. Through the hole thus made the needle of an ordinary hypodermic syringe was pushed right into the substance of the cerebral hemisphere, and about 3 cc. of pure antitetanin was injected.

The needle having been withdrawn, the wound was dressed with iodoform and cotton wool.

Throughout the operation the pony gave no indication of experiencing inconvenience; it was returned to the box and not seen by me again for twenty-four hours, at the end of which period there was a very marked abatement of the tetanic symptoms. The groom said it had emptied its manger during the night, and it was feeding fairly well when I saw it. During the next ten days the tetanic symptoms

¹ See article by MM. E. Roux and A. Borrel, "Journal of Comparative Pathology and Therapeutics," June 1898, Vol. XI., Part II.

gradually disappeared, and at the end of that time it was turned out into a paddock, having made a good recovery.

The wound in the forehead was not touched until the third day after operating, when, as it was suppurating, it was cleansed and dressed with iodoform. This was repeated when necessary, and when the pony passed from my observation on 7th August only a small scar indicated the seat of the operation.

Throughout there was no indication of any ill effect from injecting into the brain substance. The animal was a trick pony belonging to a travelling circus, and it had not forgotten its performance, so that its mental capacity did not appear to be affected.

CASE II.—On the 2nd November last a hunter received a punctured wound in the off hind heel, and on the 23rd of the same month tetanus supervened. The course of the disease was acute, and by the 26th the tetanic spasms were very severe, and trismus was very marked. On the morning of 26th, having injected 6 grains of cocaine under the skin, a hole was drilled in the parietal bone in the same way as in the case of the pony, and with my ordinary tuberculin syringe I slowly injected 8 cc. of pure antitetanin, pushing the needle into the cranial cavity to a depth of 2 inches.

Immediately after the withdrawal of the needle the horse became very violent, its limbs were convulsed, defecation occurred, and it appeared to be about to fall as though in an epileptic fit. The horse, however, supported himself against the wall, and within five minutes the severity of the convulsions had passed off, but it was impossible to dress the wound on his forehead except by throwing a little iodoform on it.

On the morning of 27th the symptoms of tetanus had considerably lessened, there being, as in the previous case, a very great improvement; the horse was feeding well, and the movements of both limbs and jaws were much less stiff.

The recovery has since been gradual, without any ill effect from the injection; the horse has gone to walking exercise daily since 8th December, and to-day (12th December) he is practically well.

He has always objected to the wound on his forehead being dressed, so that it has had very little attention; nevertheless, it has done well, and now only a small scab remains.

My experience has been that recovery from an attack of tetanus in the horse usually takes six weeks, but in both these cases the duration was scarcely three weeks, although in one a delay of nine days, and in the other of three days, was permitted before resorting to the injection.

These are the only cases in which I have made use of the intracerebral injection of antitetanin; the results are so encouraging that perhaps other veterinary surgeons will give this treatment a trial.

In some future case I propose injecting both cerebral hemispheres at an interval of twenty-four hours, anticipating that recovery will be more rapid than when a single injection only is made.

The antitetanin was kindly supplied to me by Dr George Dean of the Jenner Institute of Preventive Medicine. It was quite fresh and pure, Dr Dean being of opinion that an admixture of even a small quantity of carbolic acid would when injected into the brain produce intoxication.

CLINICAL NOTES ON AFRICAN HORSE-SICKNESS.

By J. T. COLEY, M.R.C.V.S., Vet.-Lieut., A.V.D.

THE following notes on African horse-sickness and its treatment during four seasons' experience may be of some interest to the profession.

Symptoms.—As a rule no symptoms appear before the final stage of the disease, and frequently none are observable until a few hours before death; but if the thermometer be daily used it will be found that the animal's temperature gradually rises from three to six days before any other symptoms appear. The first symptom is fever, and this is soon followed by a yellowish-pink discolouration of the conjunctiva, which is often congested, and invariably has petechial or stellate spots, or ecchymoses. A peculiar dry husky breathing can be detected on auscultation at the thorax. In a day or two these symptoms become more apparent, and are followed by acute congestion of the conjunctiva, thickening of the eyelids, and swelling of the supra-orbital fossæ. The neck is swollen along the jugular furrows, and in the "dikkop" or "big-head" form the head may become of an enormous size. The eyes are closed, the conjunctivæ protrude, and the breathing is stertorous, not unlike the symptoms seen in anthrax; blowing and heaving a short time before death; the respiration is "heaving." The animal is rarely off feed until this blowing stage, and therefore without daily use of the thermometer to detect the disease in its early stages treatment is not likely to be of much avail.

Treatment.—I have tried every treatment I could hear of, but without much success.

The only treatment with which I had any success was the intravenous injection of a solution containing 4 grains of iodine and 15 of potassic iodide in 1 drachm each of glycerine and boiled water.

This caused no inconvenience or disturbance to the animals, and on the following day a marked improvement was noticed in the temperature and conjunctivæ, especially if the latter had been much swollen or congested.

I previously used intratracheal injections of turpentine, carbolic acid, iodine, and potassic iodide in glycerine, and that also had good results, but it very often caused an undue amount of disturbance and coughing to the animal, which is all avoided by intravenous injection. I may mention that I never had any abscess or local lesion from intravenous injection, and the place could not be detected a few minutes after injection, although I have done some hundreds.

Arsenic is the only drug that I can say I found anyway successful as a preventative.

The following is a temperature chart of some cases treated intravenously, which gives an idea of the treatment, as all are typical cases of "horse-sickness," and are just copied from a few notes I chance to have by me.

Of thirty-six animals treated by intratracheal injection, twenty-three died within twenty-four hours of admittance, six died from two to six days later, and seven recovered.

Of seventy-eight treated intravenously, fifty-two recovered and twenty-six died. Fifty-seven others died on the day of admission, and about one-half of those had received one injection.

Morbid Anatomy.—The lungs are nearly always congested, and often very voluminous, with the interlobular spaces well marked by a yellowish jelly-like exudate. A similar exudate is often present under the pleura, with some patches of pleurisy. The bronchial tubes invariably contain whitish or blood-stained froth, and in many cases the trachea is full of froth and exudation, which escapes from nostrils after death, as shown in the annexed illustration. The trachea is not inflamed.



Photograph of horse dead of horse-sickness, showing the characteristic formation of white froth at the nostrils.

The pericardium in every case contains straw-coloured or blood-stained fluid, varying from a few ounces to pints.

The heart in a number of cases has extravasations, and *ante-mortem* clots on one or more of its valves.

The spleen is nearly always congested and enlarged, and the liver is in the same condition.

The congestion of the stomach noticed by Professor M'Fadyean was only seen by me in a few cases. A good many appeared normal, but this organ was not examined in every case.

The adipose tissue of the chest, along the jugular furrows, about the eyes and head, nearly always contained more or less of the straw-coloured exudate. As a rule, other organs appeared normal.

From the above notes it will be seen that treatment is not of much avail, so that to combat this disease measures of prevention must be relied upon.

I had an opportunity of studying preventive measures with Dr Edington at the Bacteriological Institute, Grahamstown, and he very

kindly showed and explained all his experiments to me. It is to be hoped that sooner or later he will be rewarded by discovering an attenuated virus which can be used by every farmer, without professional aid, as a preventive measure.

I gathered from Dr Edington that we have as yet no microscope capable of dealing with the organism of horse-sickness, nor any filter that will arrest it, and that one has thus more or less to work in the dark in attempting to obtain a proper attenuated virus suitable for protective inoculation.

HORSES KILLED AND INJURED BY ELECTRIC SHOCK.

By R. LEWIS GREEN, M.R.C.V.S., Dudley.

DURING a severe snowstorm on the 12th December many of the telegraph and telephone wires in this town broke and became entangled with the overhead trolley wire belonging to the Electric Tramway Company, and in two of the suburbs three horses were thereby killed, and four others were more or less severely injured.

I will first describe the *post-mortem* appearances of the three that were killed.

The first case was a grocer's van horse, one of a pair. Both received the shock simultaneously, owing to the wires falling on them as they were passing. Both fell to the ground instantly. This animal was between fifteen and twenty minutes dying. External injuries in the shape of wire burns were present on the inside of the near knee, both hind fetlocks, off hip, off hock, and off shoulder. The burns in this case were as if performed with a red hot skewer.

Blood was exuding from both nostrils, extreme rigour mortis was present, and the visible mucous membranes were highly injected. On removal of skin the muscles of the shoulder, ribs, flanks, and the panniculus carnosus showed lesions such as would arise from burning. The muscular tissue had a peculiar greyish-red or purple appearance. The external jugular vein was filled with blood which presented a dark appearance, tarry in colour, and of the consistency of treacle. The cutaneous vessels were congested, and over the seats of injury ruptured, and the dark-coloured blood before described was issuing from them. The walls of the heart were highly congested, and the ventricles contained about three-quarters of a pint of dark tarry blood. The brain substance was healthy, but the membranes were dark and congested. The whole depth of the convolutions and the right ventricle were blood-stained.

The other two animals killed were attached to a hearse. They came into contact with fallen wires and died instantaneously. This occurred in another district entirely, but at about the same time as the first case. The only external marks present in one of them were five wire burns on the near elbow, of no depth, just singeing the hair. The other was burnt in four or five places on the posterior part of the elbow and the anterior portion of the chest, the longest one being about 15 inches long and 1 inch deep.

The *post-mortem* appearances were with two exceptions the same as

in the first case, viz., there were blood stains in the ventricles of the brain, and the muscular tissue was not so much changed in tint.

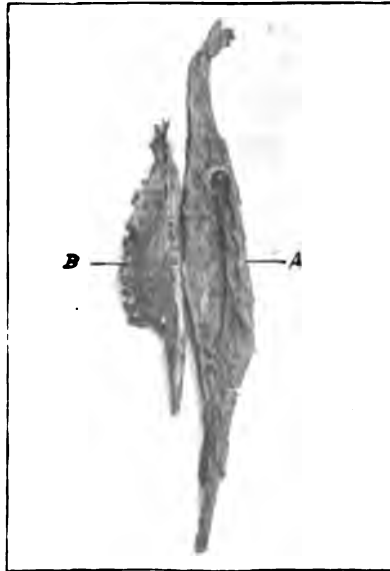
The animals killed instantaneously had no bleeding from the nostrils, while the one that was some minutes dying, and those that did not have sufficient current passed through them to cause death, bled from both nostrils. The latter animals seemed to speedily recover, and now show no ill effects from the electricity, save that one of them, an aged mare, was burnt severely, and there seems an undue delay in starting granulations. She has five burns on the off shoulder, each about 5 inches long; one on the off shoulder about $4\frac{1}{2}$ inches long and half-an-inch deep, one over the ribs about 8 inches long and 1 inch deep, and several minor ones on the off side of the chest.

COMPOUND FRACTURE OF THE SCAPULA: SUCCESSFUL REMOVAL OF THE DETACHED SPINE.

By the Same.

THIS was an interesting case, but I must describe it from memory (it only occurred last March) as I have lost my notes taken at the time.

Subject.—Chestnut cob, fifteen hands. It was being driven in a phaeton, and while turning it was run into by a horse and cart from



A. Detached spine of scapula.
B. Part of supraspinous fossa.

behind, the shaft penetrating the muscles of the shoulder and fracturing the scapula, completely detaching the latter, and leaving a gap in

the bone extending from about 2 inches above the cavity up to the cartilage of prolongation.

As the animal showed no lameness I decided to operate, and I did so the same day (5th March), enlarging the external opening for a distance of about 10 inches. The spine was pulled in a posterior direction, and held down so firmly by its muscular attachments that to elevate it, so that I might get to work with a knife, I had to place a piece of shoeing iron under it, and get a blacksmith to bear on that with some considerable pressure.

I commenced at the lower end, gradually releasing the spine from the muscles; and when the highest point was reached I had to cut about 1 inch out of the cartilage of prolongation to free it. When dissected out, it measured $9\frac{1}{2}$ inches long by about 3 inches wide. The piece of bone alongside of it in the illustration is a portion of the supraspinous surface, and it was about 4 inches by $1\frac{1}{2}$ inches; this also had to be cut away, but that was done with very little difficulty.

The gap left was greater than I anticipated, but I plugged it up with cyanide gauze, sprinkled with powdered boric acid and chinosol (1-8), inserting about three sutures to keep it in place. This was left for forty-eight hours. When removed there were no signs of suppuration, so I repeated the dressings and left them for the same length of time. Afterwards I used small plugs of gauze saturated with hydrag. perchlor. (1-1000), alternated with ac. carbol. (1-20).

In about a fortnight or three weeks I discontinued the gauze, swabbing out daily with one of the before-mentioned antiseptics, and dusting with boric acid and chinosol.

Granulation went on at a great rate, and on 15th April the animal was discharged, sound in action, but not ready to go to work owing to a small wound at the seat of puncture by the shaft. After several applications of liq. epispasticus this healed, and the animal went to work in June, a breast collar being used so that the parts might recover completely. This he wore for about six weeks, after which did his work in an ordinary collar, and he has continued at work since. The only disfigurement is a gap in the position formerly occupied by the spine.

Reports.

TRICHINOSIS IN HOME-BRED AND IMPORTED PORK.

THE following is taken from a report recently presented to a Committee of the Glasgow Town Council by Mr A. M. Trotter, M.R.C.V.S., Chief Veterinary Inspector at the Moore Street Abattoir, Glasgow:—

The investigation was undertaken to determine the prevalence of the *trichina spiralis* in home and foreign pork, and the influence of this parasite on public health.

As the object of the investigation was to determine the source of danger, and as it is established that man becomes trichinosed through the ingestion of infested pork, the enquiry was limited to an examination of this article of food. In addition to home supplies, large quantities of pork in a fresh or preserved condition are annually imported from the United States of America, Canada, Denmark, and other countries, consequently it will be advisable to consider these under their respective headings—*i.e.*, home and foreign.

Although the *trichina spiralis* was discovered in this country by Peacock in 1828, hitherto no investigation to determine whether or not trichiniasis is prevalent in our home swine has been carried out. It has been ascertained that this parasite is not uniformly distributed throughout the muscular system, but certain muscles are more liable to be infested than others. Messelneitz examined 38 trichinosed pigs during the years 1893 to 1896. From each of 10 selected parts he made 36 preparations, and found most trichinæ in the pillars of the diaphragm, and arranged in the order of frequency came the muscles of the tongue, diaphragm, larynx, abdomen, haunch, intercostal, chest, shoulder, and gigot. The work of examination is therefore facilitated, and greater accuracy obtained, by the selection of several of these muscles.

The *modus operandi* adopted in the investigation was as follows: From each carcase three pieces of muscle were removed—*i.e.*, from (1) the abdominal wall, (2) the neck, and (3) the pillars of the diaphragm. From the three pieces 24 microscopical preparations were made, and examined under a low power $\times 60$.

The result of the enquiry is extremely favourable, as not one case of trichiniasis was discovered in the 3000 home swine examined. It must not, however, be concluded that the pigs of this country are free from this parasite, as, during 1896, of 239,115 swine examined in Wiesbaden only 1 was found infested; of 206,556 in Luneburg, 3; of 209,088 in Minden, 5; of 235,661 in Hanover, 10; of 220,603 in Hildesheim, 12; of 467,667 in Magdeburg, 34; of 470,837 in Oppeln, 75; of 845,857 in Berlin, 170; of 481,327 in Breslau, 105; of 148,447 in Bromberg, 166; and of 243,844 in Posen, 663; whilst of 217,422 in Cologne, 120,122 in Stade, and 115,911 in Osnabrück were found free.

It is evident from these statistics that the disease is more prevalent in certain districts than in others, and undoubtedly it depends in a great measure on the sanitary conditions of the country.

It is reasonable to suppose that home swine presently consigned to our markets are not to any great extent, if at all, infested with the *trichina spiralis*. This supposition is corroborated by the results obtained from an investigation

into the prevalence of trichiniasis in rats. This condition is rarely met with in rats other than those having access to infested animal debris, or those located in a district where the swine are the hosts of this parasite.

Where Obtained.	Number		Per cent.
	Examined.	Affected.	
Moore Street Abattoir	197	5	2.53
Milton Street Abattoir	65	0	—
Victoria Street Abattoir	3	0	—
Hide Mart	98	3	3.06
Stables (City)	74	0	—
Stables (Ayrshire)	35	0	—
Piggery (Wigtown)	31	0	—
	<u>503</u>	<u>8</u>	<u>1.59</u>

From the above table it is seen that only in two places were the rats found infested, and the question arises as to the source of infection. In Moore Street Abattoir large numbers of swine are annually slaughtered (52,516 in 1900), but as the rats caught in this place have every access to the adjoining meat market, where large quantities of American pork loins, shoulders, and hams are sold daily, the probability is that they became infested through this medium. The Hide Mart, too, is in close proximity to shops dealing in American hams. A point of interest is the absolute freedom from trichiniasis of rats having no access to this source. It is asserted by some naturalists that the rat is the natural host of the trichina spiralis, but these results, whilst not contradicting, do not support, this contention. All that it is possible to state is that in an infested area a large number of rats become infested with the parasite, and that these animals are a source of danger through pigs eating the dead bodies of those infested.

These results compare favourably with those obtained in similar investigations; for instance, Goujon examined 72 rats caught in the sewers of Paris, and found 5, or 6.94 per cent., infested; and Fessler obtained 24 rats from the abattoir attached to the Meat Market at Bamberg, and found 12, or 50 per cent., infested. In Germany the rats caught in slaughter-houses and in knackeries were ascertained to be affected with trichiniasis to the extent of 2 or 3 per cent. and 22 per cent. respectively.

In the United States of America, however, an observer found 76.47 per cent. of the rats caught in Boston Abattoir to be infested, and similar results were obtained in those captured in a large exportation slaughter-house in the same city.

Trichiniasis of swine in America was discovered by Leidy in 1847, and since then research has shown that it is prevalent among the porcine species to an alarming extent. On 6th March 1883 the German Government prohibited the importation of American pork, and this action was followed by many of the principal European Powers. This prohibition, however, was revoked by the German Government on 3rd September 1891, and by that of France on 30th December 1891, and it is now permitted to enter these countries, but only under the most stringent regulations.

Those issued by the French authorities are as follows:—

(1) Pork from the United States of America must be imported through the ports of Dunkirk, Havre, Bordeaux, Marseilles, Boulogne, or Dieppe.

(2) Each consignment must be accompanied by a certificate from the Inspector of the United States Agricultural Department on duty at the establishment where the swine were slaughtered to the effect that the pork is perfectly sound and fit for food.

Each package must also bear the official label that the pork has been examined and passed by the said Inspector.

(3) All imported pork, before it is placed on the market, must be examined and passed by the Veterinary Surgeon attached to the French Government.

All pork pronounced unfit for human food must be destroyed.

The Government of the United States during 1891 organised a staff to examine microscopically all pork intended for export to those countries insisting on this examination, and on 15th March 1899 issued the following rules and regulations:—

A microscopic examination for trichinæ must be made of all swine products exported to countries requiring such examination.

(a) No microscopic examination will be made of hogs slaughtered for interstate trade, but this examination will be confined to those intended for the export trade, and only at abattoirs which export pork products to countries requiring a certificate from this Government to secure the admission of such meats. All Inspectors issuing certificates for export of swine products which have been microscopically examined will enter upon the same the number of each stamp used, also the name of the consigner, consignee, destination, and description of article.

(b) The purple meat inspection stamp and certificate will be used only for packages containing products of hog carcasses which have been microscopically examined for shipment to countries requiring such examination.

All stamps upon packages containing microscopically inspected meat must be placed in grooved spaces, as provided by Paragraph (a), Section 12, and covered with tin. Stamps upon barrels need not be placed in grooved spaces, providing tins having raised centres, one-eighth of an inch high, the size of stamps, are used. The tins placed over stamps must be sufficiently large to go over the whole.

All stamps upon other packages shall be arranged as provided by Section 12.

21. The microscopic inspection of pork intended for export to countries requiring such inspection shall be conducted as follows:—

When the slaughtered hog is passed into the cooling room of said establishment, the Inspector in charge, or his assistants, will take from each carcass three samples of muscle—one from the “pillar of the diaphragm,” one from the psoas muscle, and the other from the inner aspect of the shoulder, and also from the base of the tongue, when that organ is retained for exportation; and said samples will be placed in small tin boxes, and a numbered tag will be placed upon the carcass from which samples have been taken, and a duplicate of said tag will be placed in the box with said samples. The small boxes will be placed in a large tin box provided with a lock. The boxes containing the samples from the hogs in the cooling-room so tagged will be taken to the microscopist for such establishment, who shall thereupon cause a microscopic examination of the contents of each box containing samples to be made, and shall furnish a written report to the Inspector giving the result of said microscopic examination, together with the numbers of all the carcasses affected with trichinæ.

The samples of pork microscopically examined shall be classified as follows:—

Class A.—Samples in which there are no signs of trichinæ, living or dead, calcified cysts, or other bodies or substances having any resemblance to trichinæ or trichinæ cysts.

Class B.—Samples in which there are disintegrated trichinæ or trichinæ cysts, calcified trichinæ or trichinæ cysts, or bodies having any resemblance thereto.

Class C.—Samples in which there are living or dead trichinæ bodies not disintegrated.

22. All carcases reported by the microscopist to the Inspector as affected with trichinæ (Class C) shall be removed from the cooling-room of said establishment under the supervision of said Inspector, or that of some other reliable employee of the Department of Agriculture, and shall be disposed of in accordance with the provisions of Section 7, or they may be rendered into edible lard at a temperature of not less than 150° F., or made into cooked meat products if the temperature is raised to the boiling point a sufficient time to thoroughly cook the interior of the pieces.

Carcases belonging to Class B shall be rejected for shipment to countries requiring microscopic examination. All meat belonging to Class C, and which is to be cured before being cooked, shall be marked with twine and seals provided for that purpose.

23. Separate cellars must be provided in which microscopically inspected meat must be cured and stored, and no other meats are to be allowed therein. These cellars must be so arranged that they can be securely locked, and the key must remain in the possession of a trusted employee of the Department, who must lock and unlock the cellar as the business requires. He must also keep an exact account of all meats going into and from the cellar.

(a) Before the cutting up of microscopically inspected carcases is commenced all carcases which have either been condemned or rejected must be removed from the rail upon which the passed carcases are hanging, and after the passed carcases have been cut up and disposed of the condemned and rejected ones must be disposed of in accordance with these regulations.

The cutting up of carcases which have been passed must be done under the personal supervision of an employee of the Department. Before cutting is commenced all benches, chutes, etc., must be cleaned of all other meats. When it is finished a stop must be made to clear away all the meats before the cutting of other hogs is begun.

(b) The greatest diligence must be exercised at abattoirs where sausage, brawn, and others products of a similar nature are prepared from microscopically inspected meat and the same are handled. Such sausage must be kept in separate compartments provided with locks, the same as other microscopic meats, and must also be prepared in separate rooms and cut up in choppers used only for such sausage. An absolutely correct record must be kept by weight of all such meats, in order to prevent any but passed products being used.

(c) Whenever it is necessary to remove hams, shoulders, sausage, or other microscopically inspected meats from their respective compartments for smoking or other preparation, a separate smokehouse or compartment must be provided, or each piece must be marked by seals affixed by an employee of this Department.

24. At all cities where inspection is established Inspectors are requested to notify the municipal authorities of the character of the inspection, and to co-operate with them in preventing the entry of condemned animals or their products into the local market.

The amount of pork microscopically examined during the fiscal years 1892 to 1899 is here shown :—

	To Countries Requiring Inspection.	To Countries not Requiring Inspection.	Total.
1892 .	22,025,698 lbs.	16,127,176 lbs.	38,152,874 lbs.
1893 .	8,059,758 "	12,617,652 "	20,677,410 "
1894 .	18,845,119 "	16,592,818 "	35,437,937 "
1895 .	39,355,230 "	5,739,368 "	45,094,598 "
1896 .	21,497,321 "	1,403,559 "	22,900,880 "
1897 .	42,570,572 "	1,001,783 "	43,572,355 "
1898 .	120,110,356 "	161,303 "	120,271,659 "
1899 .	108,858,149 "	70,046 "	108,928,195 "

The second column, showing the amount of pork microscopically examined consigned to countries not exacting this inspection, is of interest, as it shows that year by year, through the indifference and apathy of their respective Governments, this protection to public health is a diminishing quantity. It is candidly explained in the Annual Report of the Bureau of Animal Industry (United States of America) for 1896, p. 24, that "the shipment of microscopically inspected pork to countries not requiring this inspection has been intentionally discouraged, as the expense in such case is not warranted."

The cost of the microscopic examination varies from year to year, and is defrayed out of the public funds, but it has been suggested that it could be more legitimately assessed against the trade which it benefits. During the year 1899 the cost of this inspection was 19,835,514 dollars, an average of 8·9 cents for each carcase examined and 0·182 cent. for each pound exported. The cost of inspection is increased, however, by the examination of small pieces, as these require as much time as is necessary for a whole carcase, consequently it was found unavoidable to restrict the weight of individual pieces to five pounds and upwards.

The result of the microscopic examination for 1897, 1898, and 1899 is here detailed:—

	EXAMINED.		INFESTED.			
	Carcases	Pieces.	Carcases.	Per Cent.	Pieces.	Per Cent.
1897 .	550,291	1,331,018	3,243	·58	10,082	·75
1898 .	1,927,838	875,008	35,707	1·85	10,966	1·25
1899 .	2,227,740	—	67,510	3·03	—	—

Mr Trotter found it impracticable to obtain a large number of specimens of American pork, but of 47 shoulder cuts destroyed on account of decomposition, 1, or 2·12 per cent., was infested with living trichinæ.

No recent statistics are available as to the prevalence of this condition in Danish swine, but Neumann, in his work on "Parasites and Parasitic Diseases of Domesticated Animals," mentions that at Copenhagen the proportion of trichinosed pigs was 12·15 per 1000.

Numerous experiments have shown that even in preserved pork the vitality of trichinæ is not necessarily impaired, consequently its uncontrolled importation constitutes a danger to public health.

The official records obtained from the Principal of the Statistical Office of H.M. Customs show that during 1900 8,388,031 cwts., or 419,401½ tons of pork, bacon, and ham were imported into the United Kingdom from the various sources of supply. If of this quantity only 1 per cent. was trichinosed, then it follows that 83,880 cwts., or 4194 tons of diseased pork were permitted to be imported into this country and sold for human food. It is questionable if anyone would willingly partake of pork infested with the embryos of the trichina spiralis, even if he or she was assured that the vitality of these parasites is impaired by prolonged cooking. The importation of trichinosed pork is not only a direct but it is also an indirect menace to public health, as it is a source of danger to the swine of this country. Pigs fed on kitchen slops containing pork infested with living trichinæ will certainly become the host of these parasites, and one can readily conceive of such circumstances promoting the spread of trichiniasis, which might ultimately lead to an extreme prevalence of this most dangerous form of parasitism.

The author suggests that means be adopted to prevent the importation of pork, bacon, and ham infested with trichinæ, and to attain this object that Government be approached and requested to institute a thorough microscopic examination of all such consignments for the detection of this condition.

ROYAL VETERINARY COLLEGE, LONDON.**INAUGURATION OF THE WINTER SESSION, 1901-1902.**

THE Winter Session of this Institution was opened on Tuesday, the 1st October last, when the inaugural address was delivered by Mr EDGAR M. CROOKSHANK, M.B., Emeritus Professor of Comparative Pathology and Bacteriology, and Fellow of King's College, London. Mr ALBERT BRASSEY, M.P., presided, and, in addition to the members of the staff and a number of Governors, there was a large attendance of students and members of the profession.

The CHAIRMAN, who upon rising to open the proceedings was warmly greeted, said he had the pleasing duty of occupying the post of Chairman on that occasion, and he was happy to think it was not a very arduous duty. He was very pleased to see so many students there, and he only wished there had been more. It was customary on these occasions to have an introductory address, and Professor CROOKSHANK had taken the trouble to come all the way down from Scotland, some 600 miles, in order to deliver the introductory address to them that day.

Professor CROOKSHANK then delivered the following address:—

"GENTLEMEN,—I esteem it a great honour to have been invited to deliver the introductory address, and it is a sincere pleasure to me as a Governor of this College to welcome to-day both new and old students. It is very gratifying to all those who have the interests of this College at heart, that the number of students for several years has been so well maintained. I congratulate the new students upon the choice they have made in selecting the veterinary profession as their career in life. I congratulate them also upon the advantages which they will derive as students of this Institution.

"The Royal Veterinary College not only stands in the front rank of veterinary schools in the British Empire, but it compares very favourably with those great institutions in France and Germany which have a world-wide and long established reputation.

"In this country great difficulties have to be encountered by those who desire to promote scientific education. There is not the same amount of sympathy and financial support which is given to scientific institutions in America and on the Continent. And therefore I feel very strongly that those who are responsible for the management of this College may, considering their limited resources, claim great credit for making the teaching so efficient, and for placing opportunities for original research within the reach of both the teaching staff and the students.

"With very many parents it is a most difficult matter to choose a career for their sons, and therefore it ought to be more widely known that it is a great advantage for young men to pass through a course of training in a veterinary college. Much might be said with regard to the danger of overcrowding the profession, but I venture to think that there is still plenty of room. The ranks of those engaged in private practice have to be filled up. There is a great demand in the Army for thoroughly trained veterinary surgeons. Many members of the profession have rendered splendid services in the great war in South Africa, and if the experience of this war leads to the employment of mounted troops in still greater numbers, if, in other words, horses are destined in future wars to play such a conspicuous part, there must

necessarily be a still greater demand for the services of those who have to select and take charge of them. There is also an increasing demand for veterinary surgeons in the Colonies. In South Africa and our other possessions the best methods of stamping out animal plagues must occupy the attention of the Colonial Governments, and thus give employment to veterinary surgeons.

"There must also arise a greater demand at home for their services. The eradication or diminution of such diseases as bovine tuberculosis could only be attempted by the intelligent co-operation of the public with veterinary officers of health.

"There are many parents who send their sons to the universities without having definitely settled upon their future career, and many parents who could not aspire to sending their sons to a university would do well to secure the advantages of a scientific education by sending them to a veterinary college. They would receive a sound scientific training, and the fees for the whole curriculum, exclusive of the cost of living, are less than the fees for sending a boy for a year as a boarder to one of the best preparatory schools. If the student did not eventually practise as a veterinary surgeon, the time spent in acquiring veterinary knowledge would be by no means lost. Every well-educated man should have some acquaintance with the subjects which are taught at a veterinary college, such as botany, biology, physiology, chemistry, and even anatomy. Anatomy is of special value in training the intellect. It is an exact science, and the mind of the student is not disturbed by new theories and a variety of opinions. It encourages accuracy in observation, it necessitates an accurate memory, and it trains the hands in delicate operations. As the student passes on to the essentially professional subjects, I mean the recognition, cure, and prevention of the diseases of animals, especially of horses or cattle, it will at once be admitted that there are many occupations in which a knowledge of these subjects can be put to practical account. A student may not ultimately elect to practice, he may prefer to take up farming either at home or in the Colonies. He may become an estate agent, or be called upon to lead the life of a country gentleman, and perhaps serve on the "Diseases of Animals Committee" of a County Council, or other local governing body. In any of these positions the information he would have received would prove to be of very great advantage.

PRELIMINARY EDUCATION.

"In the education of students before they come to the College, it is very desirable that more attention should be given to foreign languages, especially French and German. Foreign veterinary surgeons have a great deal to learn from our methods, and English veterinary surgeons are equally indebted to the work and experience of their foreign colleagues. Some of the best textbooks were written by foreign authors, and if the scientific veterinary surgeon in England wishes to keep in touch with Continental opinion, experience, and research work, it is an enormous advantage to be able to read the original publications. To depend upon the chance of translation, or upon meagre abstracts in English is very unsatisfactory, and many contributions to literature would be quite out of reach. Those who hold office as advisers to County Councils, or aspire to appointments in the Veterinary Department of the Board of Agriculture, would appreciate the value of being able to study the Reports of the Agricultural Departments of other nations.

"The admirable Reports of our own Board of Agriculture would at a glance illustrate this point. This is, however, not the only value of a knowledge of French and German. International veterinary and science

congresses are becoming more and more popular. The opportunities for exchanging opinions and experience with other nations are increasing, and the hard-worked veterinary surgeon might combine with the enjoyment of travelling the opportunity for exchanging ideas with his professional brethren by attending these congresses in foreign countries.

"I do not require to be told that the veterinary surgeon has to think first of making a living, I am quite aware of that fact; but it must be remembered that travelling on the Continent is a very different matter from what it was twenty years ago. The expenses have been reduced to such an extent by co-operation that an opportunity for foreign travel is placed almost within the reach of all. I would not even leave out the students; I should like to see parents encouraging the idea of 'Student's Co-operative Tours,' thus enabling them to combine a little sight-seeing and the enjoyment of travelling, with a visit to the museums and laboratories and school buildings of such institutions as the great Veterinary School of Alfort, near Paris, the Veterinary School at Berlin; and the Pasteur Institute in Paris, and the Hygienic Institute at Berlin, where diseases common to man and the lower animals are constantly the subjects of investigation. Travelling scholarships would be of quite as much advantage to veterinary as to medical students.

HIGHER EDUCATION.

"I do not propose to discuss the question of the higher education of veterinary students, but I trust I may be forgiven if I again allude to a subject which I referred to on a similar occasion many years ago—I mean the desirability of conferring a degree in Veterinary Science. I desire to draw the attention of the leaders of your profession to this question—Why should not the Veterinary College be recognised in the New University of London? I should also again point out what a great advantage it would be both to veterinary surgeons and to the public if the Royal College of Veterinary Surgeons could see their way to instituting a special examination and conferring a Diploma of Preventive Veterinary Medicine. The course of training should be somewhat similar to the course of instruction given to medical men for the Diploma of Public Health. I hope to see the day when every county council and every rural and urban council will appoint a Veterinary Officer of Health, and every applicant for such a post be required to hold the Diploma of Preventive Veterinary Medicine.

"I hope that in the future more veterinary surgeons will be placed in a position which will enable them to devote their lives to original research. There is an enormous field for investigation in the causation and prevention of diseases of animals.

TUBERCULOSIS.

"It would be impossible to give a better illustration of this fact than by referring to tuberculosis in animals, especially cattle. At the recent International Congress in London the relation of bovine tuberculosis to human tuberculosis was prominently brought forward in a paper read by Dr Koch, and the question is of so much importance that the Government has appointed another Royal Commission to enquire into it. The Governors, the Staff, and the students of this College are all, I am sure, very proud of the fact that the researches made by your distinguished Principal and the great reputation which he has made by his knowledge of comparative pathology have led to his selection as a member of this most important Commission.

"It is instructive and encouraging to the veterinary profession to know that the researches which created most interest at the Congress were those under-

taken in different Veterinary Colleges. Dr Koch's experiments were carried out during the past two years with the co-operation of Professor Schütz in the Veterinary College at Berlin. It is necessary to refer to these researches in some detail to explain the points at issue and to compare the work and conclusions of Dr Koch with the experiments and opinions of others who have investigated this subject. Dr Koch in various ways inoculated nineteen cattle with human tubercular virus and none of them showed any symptoms of disease. On the other hand, cattle inoculated with bovine tuberculosis suffered, without exception, the severest tubercular disorders of the internal organs. Dr Koch concluded that human tuberculosis differed from bovine, and could not be transmitted to cattle, and he further announced that whether man was susceptible to bovine tuberculosis at all was not yet absolutely decided, and that if such susceptibility really existed the infection of human beings was but a very rare occurrence. Dr Koch believed that the extent of the infection by the milk and meat of tuberculous cattle and the butter made of their milk was hardly greater than that of hereditary transmission, and therefore he did not deem it advisable to take any measures against it. I must express myself in full agreement with Professor Koch that if infection occurs at all, it is of very rare occurrence. But I entirely disagree with the statement that human tuberculosis cannot be inoculated in cattle, and I must add that I consider Dr Koch's statement with regard to preventive measures as singularly unfortunate. It conveys the impression, as pointed out by Dr Hueppe, that Dr Koch would have us concede to dairymen and milksellers the right of selling tuberculous milk. I feel justified in disagreeing with Dr Koch on these points because in the course of an enquiry on "Tuberculosis in relation to the Public Milk Supply," which was published in the Report of the Board of Agriculture for 1888, I made the following experiment:—A perfectly healthy calf was inoculated intra-peritoneally with very virulent human tuberculous sputum and the result was extensive tuberculous deposit at the point of inoculation with hundreds of tuberculous new growths extending from it. The calf died of blood poisoning forty-two days after inoculation and sufficient time had elapsed for the most pronounced infection of the peritoneal cavity. On microscopical examination extremely minute tubercles were found disseminated throughout the lungs and liver. Long and beaded bacilli of the human type were found in these organs and in the peritoneal deposits. This experiment was also made in a Veterinary College. It was made in the Institution in which we meet to-day, thanks to the great interest taken in the investigation by your former Principal, Sir George Brown. There was no need for me to repeat this experiment as it was absolutely conclusive, and it does not require any Royal Commission to verify the result. Other investigators in England and America have already confirmed this experiment. Dr Sidney Martin in experiments on behalf of the Royal Commission on Tuberculosis succeeded in infecting cattle with human tuberculous virus.

"In experiment I., four calves received with their food sputum from two cases of pulmonary tuberculosis in man. One calf killed in about four weeks showed 53 nodules of tubercle in the small intestine. The second killed in eight weeks showed 63 nodules, the third killed in about twelve weeks showed 13 nodules. In the fourth calf there were no nodules at all. In calf three the nodules in the intestine contained tubercle bacilli. They were absent in the microscopical specimens made of the nodules in calves one and two, but Dr Martin pointed out that these nodules were tuberculous although no bacilli were found. In experiment II. two calves received at one feeding tuberculous sputum. In one calf killed in eight weeks there were 13 tuberculous nodules in the small intestine, and tuberculosis of the mesenteric glands. Tubercle

bacilli were found in both sets of lesions. The second calf killed in about nineteen weeks showed no disease.

"Dr Ravenel of the Veterinary Department of the University of Pennsylvania has recently carried out experiments of an equally positive character which he made known at the Congress on Tuberculosis in London. Four calves were, as in my experiment, inoculated intra-peritoneally with tubercular sputum. In one case the result was negative, in the other three a *post-mortem* examination showed that all had become infected with tuberculosis, the lesions in two being quite extensive. On the other hand, calves fed on human tubercular sputum showed no trace of the disease when they were killed for examination.

"It is not impossible to explain these contradictory results, they are quite in harmony with the view which I hold that human and bovine tuberculosis are distinct varieties of the same disease. Man is not the natural soil of bovine tuberculosis. The attempts to transmit human tubercle to cattle would not be uniformly successful, and experiments which did not entail the direct insertion of the virus into the tissues might fail entirely. It is quite possible that in my successful experimental case the simultaneous production of blood-poisoning diminished the natural resistance of the tissues and rendered the animal markedly susceptible to infection with the virus from a foreign soil. The difference which may exist in the nature of the soil upon which a virus is inoculated must always be remembered. It is well illustrated by the result obtained by the inoculation of human small-pox upon cattle. Cattle do not naturally suffer from small-pox, which is essentially a disease of man, but it can be grafted on bovine tissues in exceptional cases. These experiments are so difficult that many experimenters, who have failed, have refused to believe in the positive results obtained by others. But variolation of the cow is nevertheless possible, and so marked is the effect of the soil, that the highly-infectious small-pox of man is transformed into a locally-inoculable, mild, vesicular malady in cattle, and can never again acquire an infectious character. Sheep-pox may be given as another instance. In this case a highly infectious malady in sheep when successfully grafted on human tissues is deprived of its infectious character and reduced to the condition of a mild vesicular disorder.

"All tubercle bacilli can be inhaled and taken with food by perfectly healthy individuals without producing tuberculosis, but if from any predisposing cause there is vulnerability of the tissues, it is reasonable to be suspicious of bovine bacilli. As human tubercular virus can be grafted on to the cow, so also there are cases in which bovine bacilli invade the human tissues. I refer to those cases in which there has been direct inoculation of man. Cooks, grooms, butchers may suffer from tubercular nodules in the skin containing tubercular bacilli, but they undergo caseation and disappear.

"I agree with Dr Koch in the opinion that human infection can only be quite exceptional. If it were not so, owing to the frequency with which tubercle bacilli occur in milk, cream, butter, cheese, and the quantity of meat derived from tuberculous cows, the inhabitants of every country in the world in which bovine tuberculosis is prevalent must necessarily have been decimated by tubercular disease. I do not accept the theory that abdominal tuberculosis in children is due to infection from tuberculous milk. Those who advocate this view would appear to set aside the opportunities for infection from a human source. If tubercle in children is the result of infection of the digestive tract, there are plenty of opportunities for self-infection when there is concurrent disease of the lungs, and there are obviously many ways by which a child might be infected by the mouth with bacilli from a human source. Physicians who have had enormous experience with tubercular

patients of all ages are by no means ready to attribute consumption in any form to tuberculous milk or meat. Sir Douglas Powell in his evidence before the Royal Commission, when asked if any danger existed in consuming meat bought in the butcher's shop or in using milk from the milk-pail—*i.e.*, whether the consumption of milk and meat from tuberculous animals might cause the disease in man—replied that he had not met with any cases in his experience which would connect the two facts together. Dr Goodhart, consulting physician to the Evelina Hospital for Children, gave similar evidence. I am convinced that any suspicion of danger can be removed without creating a public scare. It is simply a question of better inspection of dairies, and any risk which may exist can be met by adopting the precaution in both private and public dairies of destroying "wasters" or "piners" and removing all cows suffering from any disease of the udder. There is no necessity to insist on the boiling of all milk. Unless there are urgent reasons it is not a practice likely to be generally adopted. Boiled milk is very unpalatable to many people, and the boiling of milk alters its composition and renders it, in the opinion of many, a danger to infants. Sir Douglas Powell and Dr Goodhart were not prepared to recommend the boiling of milk as a precaution against tuberculosis. Tuberculosis of the bowels is almost unknown in very young children, and it is not even very common in children from five to ten years. In Dr Goodhart's experience cases occurring in children with a distinct family history of tubercle were very widely spread, and it was quite common to find children becoming tuberculous after measles, bronchial pneumonia, whooping cough, and intestinal catarrh.

"As regards any danger from tuberculous meat, it is, in my opinion, practically *nil*. I do not believe there has been a single case recorded of tuberculosis contracted by eating tuberculous meat. Jews have a very thorough system of meat inspection, and yet they are by no means free from tuberculosis. When travelling in the West Indies I found that tuberculosis was by no means uncommon among negroes, and Dr Williams of Demerara stated before the Commission that Hindoos and negroes suffered severely. Dr Williams said that the Hindoos eat very little meat of any kind. The negroes also eat meat in very small quantities, either salt beef or salt pork imported from America, and this is well cooked before it is eaten. They lived, for the most part, in small and badly ventilated buildings. Negro children suffered from tubercle. They did not have as much milk as they ought, and usually, owing to the climate, the milk was boiled.

"I think we are justified in concluding that if the carcase is well nourished the meat is perfectly wholesome in spite of the existence of local deposits of tubercle in the organs and glands, which should of course be condemned. The view that an animal in prime condition but with a minute tubercular nodule was a diseased animal, and the carcase ought therefore to be destroyed, was a very extreme view, and could not be carried into practice. As a matter of fact there could be no justification for wholesale destruction of such valuable food. Compulsory destruction of every animal with a tubercular deposit would almost involve the ruin of the agricultural industry, and it would be quite inconsistent with the continued use of meat imported from countries where such drastic measures were not entertained. No government would face the question of compensation for every case of tuberculosis, however slight the lesion. It would be quite as reasonable to demand the destruction of the carcase of every animal suffering from an actinomycotic tumour. Actinomycosis is not only prevalent in cattle, but, like tuberculosis, it is also a disease of the human subject. In this country, in America, and in Australia it is still commonly known as "cancer." Cancer of the tongue, cancer of the jaw, cancerous polypus, cancerous sores, are different manifestations of

actinomycosis. Quite recently a correspondent in the *Times* has suggested that eating the flesh of such animals may account for the prevalence of cancer in man—a most fallacious suggestion, for actinomycosis and cancer have nothing in common except the popular and misleading designation. It was at one time thought, as in the case of tuberculosis, that the disease in man is derived from cattle, and I have succeeded in grafting the human disease in a calf. They are, however, in my opinion, distinct varieties, and I do not accept the theory that men and animals infect each other, but I believe they contract the disease quite independently, and that the micro-organism is probably derived from cereals. Does the tubercle bacillus, like the streptothrix actinomyces, exist outside the animal body? There are many other questions bearing on the life history of the tubercle bacillus, its exact rôle in tuberculosis, and its relation to other organisms which are closely allied to it both as regards morphology, staining reaction, and cultivation, such as the bacillus of avian tuberculosis, the tubercle bacilli obtained from fish and worms, the bacillus of Rabinowitsch from gangrene of the lung, of Marpmann from urine, of Mironescu from human fæces, of Karlinski from nasal secretion, of Rabinowitsch from milk and butter, and of Moeller from timothy grass and the dust of other grasses. It is to be hoped that, incidentally, the new Royal Commission will throw some light on these points.

“To eradicate tuberculosis in cattle and so to ensure the breeding of perfectly healthy stock, and thus to restore the confidence of the public in the supply of wholesome meat and milk, will be a splendid work for veterinary surgeons to undertake, and one to which they must direct all their energies. As regards the prevention of tuberculosis in man, that must be left principally to the sanitary inspector and the medical officer of health. We must not concentrate all our energies upon the destruction of tubercle bacilli, but rather give more attention to other factors responsible for the causation of tuberculosis. The study of tuberculosis in animals may throw some light on these causes, and experience of the disease in man may assist in elucidating the causes of disease in animals. Tuberculosis is peculiarly liable to occur among birds and animals kept in captivity. Poultry and guinea-fowls are often the subjects of this disease, and ostriches and other birds in the Zoological Gardens become infected. In monkeys in captivity the disease occasionally occurs in an epidemic form. Pheasants in preserves are attacked sometimes in large numbers, and rabbits in overcrowded warrens. These instances help to illustrate the view that damp and other insanitary conditions, confinement, overcrowding, defective ventilation, heredity, breeding in and in, were the most powerful factors in rendering the tissues prone to tubercle and a fitting soil for the invasion of the bacilli. We must not omit in the case of man the influence of alcoholism, which has been so closely investigated by Dr Brouardel; the influence of previous illnesses, such as measles and whooping-cough, as urged by Dr Goodhart; and, lastly, the influence of special trade occupations which involve inhalation of dust of various kinds.

“There are two points which I will discuss at greater length, heredity and tuberculosis in children. Heredity is of two kinds, there is hereditary predisposition to the disease, and hereditary transmission of the virus. There is an inherited susceptibility or weakness which renders many individuals liable to the development of tubercular disease. Family history plays a very important part in human tuberculosis. Sir Douglas Powell states from his experience that 48 per cent. of the cases in hospital suffering from tuberculosis had a previous history of hereditary tuberculosis. Whether all these cases are cases of hereditary predisposition, or hereditary transmission of the virus, or examples of both is not determined. I am entirely in agreement with Dr Klein and Professor Horsley that in some cases there is direct transmission of the virus

and that it may exist for many years in a latent form. Dr Klein brought this matter very convincingly before the Royal Commission. In connection with this question of heredity some interesting observations upon tuberculosis in birds have been recorded by Dr Baumgarten. On a poultry farm a cock developed tuberculosis. All the chickens reared from this cock were tuberculous. There was no evidence that the fowls were infected with either human or bovine tubercular virus. An identical case occurred on another farm, and these cases have been accepted as evidence of the direct transmission of the virus from the parent bird. Tuberculosis is a rare disease in calves. It seems probable that those cases which do occasionally occur are mostly if not entirely the result of hereditary transmission. In discussing the occurrence of tuberculosis in children we may derive some consolation from the immunity of calves. Tuberculosis in children is largely a disease of the poor. It attacks all classes, but is extremely common among the London poor and the poor of our overcrowded cities. I would attribute the disease in children to a want of milk rather than the possible occurrence of a few stray bovine bacilli in milk. Plenty of milk, good nourishing food, and better hygienic surroundings will do more to diminish the numbers of tuberculous children than any legislation directed against bovine bacilli. In proportion as the slums are removed from our overcrowded cities and the problem of the better housing of the poor has been solved we may expect to see a steady diminution in the amount of tuberculosis in children. I believe that the infectious nature of the disease has been exaggerated. It is quite possible that the theory is fallacious. I mean the idea that phthisis can be caught like scarlet fever, or may be compared to typhoid fever is, I think a mistake. In typhoid fever epidemics at home, in India, and in South Africa, we know that apart from cases of individual insusceptibility we have not to consider any other factors except the introduction of the specific poison of typhoid fever. Those in health and out of health fall victims to the disease if they have taken the poison in water or food. There is no question at all of hereditary transmission or of predisposing conditions which render the subject susceptible to the poison. At the same time the habit of spitting in public places and railway carriages and other conveyances should be prohibited, and the sputum of phthisical persons should be disinfected. Several years ago I carried out experiments proving that sputum treated with 20 per cent. carbolic acid was rendered quite inert. Guinea-pigs inoculated with the carbolised sputum remained perfectly healthy, and all control animals were infected without exception. There is no need to create a panic or raise an outcry for legislation to make spitting in public places a matter to be dealt with in the Police Court. That the virus of tubercle scattered far and wide becomes a constant source of danger to all who inhale the air is not a theory which is supported by experiment and experience. Dr Ransome maintains that in a well ventilated room it is rendered harmless. Tubercular sputum was exposed in the ventilating shaft of a hospital and proved virulent to rodents, but similar sputum exposed for the same time in Dr Ransome's room, which was ventilated and well lighted, was absolutely harmless.

"Dr Klein placed a number of guinea-pigs in cages in the ventilating shaft of the Brompton Hospital very many years ago. They were kept there for several weeks and all of them became tuberculous. We must not too hastily draw conclusions from experiments upon small animals such as rodents. In the same institution it was found among all those who had been connected with it: nurses, porters, physicians, surgeons, and so on, the mortality from consumption was quite within the average of ordinary mortality. I am convinced that if tuberculosis were readily conveyed from person to person, that marriage of individuals who become or are consumptive would be a fruitful source of direct infection. We should hear constantly of instances in which married people

had infected each other with tuberculosis owing to the opportunities which occur for conveying the disease by the breath and in other ways. It is necessary to lay great stress upon the difference between experimental inoculation and natural infection. I cannot therefore regard tuberculosis as catching in the same sense that scarlet fever is catching, nor can I regard the comparison to typhoid fever as one that is not very misleading. Those who aim at eliminating the conditions which I have referred to as powerful factors in the production of tuberculosis will render the greatest services in the crusade against tuberculosis in animals as well as in man. I trust that many of you present to-day will assist in this great work."

The CHAIRMAN said he was sure it was the wish of those present that he should express their grateful thanks to Professor Crookshank for the exceedingly eloquent and interesting address which they had just heard. The lecturer had indicated a very large scope of study for all the students. He was quite sure it was a great source of congratulation to the College that their Principal should have been elected to serve on the Royal Commission on Tuberculosis. It was not only a great honour to himself, but also a great honour to their College.

He fully endorsed all that Professor Crookshank said at the commencement of his address regarding the great importance of, and the large demand there was for, skilled veterinary surgeons throughout the country, and he felt sure that none of the students would consider the time he spent in that College as wasted time. He only wished he had had in his early days the time to become a student there, and to have had the advantages and opportunities which that College afforded, because he had been for a great number of years a Master of Hounds, and, in addition, he had constantly a large stud of horses under his charge. That, he need scarcely say, had caused him a great deal of anxiety from time to time. He remembered once when he was in Ireland being informed that fifteen horses composing the stud were laid up with influenza. That was not a very enviable position for a Master of Hounds to be in. But what did he do? He immediately applied to the Royal Veterinary College, and they promptly sent down a professor, to whose skill it was entirely due that they soon checked the disease and prevented it from spreading. On another occasion a number of the hounds were attacked with warts in their mouths, and one of the College professors was consulted. He soon brought them round, and prescribed a treatment which was adopted, and which resulted in their having no other bad cases. They all recovered after a time.

That only showed how important it was, particularly to a Master of Hounds, that he should know something about veterinary science. He hoped that all of them would devote all their efforts to making themselves masters of the profession, and that they would be successful in after life.

He begged to move a vote of thanks to Professor Crookshank for his most eloquent address. (Applause).

The vote was passed by the students in their customary hearty manner.

Professor CROOKSHANK, who was loudly cheered in acknowledging the compliment, said he thanked them very much for this cordial vote of thanks. It had been for many years, and he hoped it would be for many years to come, his privilege to do what he could for the Royal Veterinary College.

He was afraid that he had treated the new students to some of the dry pieces of pathology, and he would like to say one or two words to them in rather a different fashion. He had come down from his temporary home in Scotland in order to deliver the address to them on that occasion, and he should like to relate to them a little incident which occurred during his stay there. He was fishing in a loch which was very famous for sea-trout and

salmon, and having a very favourable day anticipated very good sport. Well, he whipped that loch the whole morning, and having had only one or two very unsatisfactory rises he must confess that he became somewhat disheartened. So he turned to his old gillie and asked him the secret of catching fish in that loch. He said, "It's just this way, sir. You never know when the fish are going to rise. They may rise either at a very early hour in the morning, or in the middle of the day, and sometimes it may not be until the evening. But there is only one way of catching fish, and that is sticking to it" (Laughter and applause).

Well, he took his advice, and a little later in the afternoon the atmospheric conditions being perhaps more favourable, first of all there was a salmon secured, then two or three large trout, then a 3 lb. trout, and finally three or four more salmon. (Laughter.) He had a moral in that story. He looked at these beautiful fish at the bottom of the boat, and he supposed he had rather an air of self-satisfaction at the manner in which his efforts had been rewarded. He thought his old gillie must have read his feelings, for he said: "The real secret you see, sir, is sticking to it."

Well, he hoped that those new students who had joined that day would carry out the good advice of the old gillie, and that in their career in after life they would gain success by "sticking to it." (Applause.) They must not get disheartened, as he admitted he had been, for their opportunity in life was certain to come. As the fishes rise so would their opportunity come. When it did they must seize it and stick well to the work that they had in hand.

Professor M'FADYEAN, at the request of the chairman, read the Prize List for the previous session (which has appeared in the preceding number of the *Journal*). He said: Professor Crookshank, to whom they were all very much indebted, had saved him the trouble of offering to the students any good advice, or of pointing out to them how success was to be attained, not only in the various class competitions, which were after all of comparatively trifling importance, but also in their career in after life. He was only going to repeat the advice in a slightly different form. Like Professor Crookshank he had occasionally fished for salmon and sea-trout, and he remembered very well that when he was getting elementary instruction in that very interesting art he used to ask everybody from whom he thought he could learn anything what was the best way to catch salmon. The best answer which he ever got was: "Keep the fly in the water" (Laughter.) He believed that advice was strictly applicable to their course of study there; in other words, they must be diligent throughout the whole course, and they must begin immediately. They would all be expected to begin their studies on the following day, not as if it were the second day of the session, but quite the same as if it were the fiftieth day. The lectures would begin at nine o'clock next morning, and he hoped the clinical work would proceed at ten o'clock. Subjects were ready for dissection, and Mr Wild would be very pleased to arrange with those who desired to begin that afternoon. (Laughter and applause.)

SIR GEORGE BROWN said he had a very easy and short task to perform to finish the interesting work of that day. They had had as Chairman Mr Albert Brassey, M.P., who had conducted the meeting to their satisfaction he had not the slightest doubt (cheers). It was an encouraging sign of the interest evinced in the veterinary profession, and it must be very gratifying to them to know that so many gentlemen in different walks of life did not scruple to devote their time, not only to occasional attendances there for the purpose of filling the Chair, but also as hard-working members on the Governors' Board (cheers). He was perfectly sure that they would allow him to propose a vote of thanks to Mr Brassey, and that they would convey their idea as to the desira-

bility of so doing in their usual emphatic way. The motion was carried with acclamation.

The CHAIRMAN, in replying to the vote, said it had been a very great pleasure to him to come there that day, and to receive such a hearty vote of thanks from them. Anything he had been able to do in acting as one of the Governors he had done with pleasure. He thought it was a matter of great importance to have a sufficient number of veterinary surgeons throughout the country. There was no doubt that there was a very great demand not only at home, but also in the Colonies and in the army for skilled veterinary surgeons, and he wished to one and all of the students success throughout their professional career (applause).

The proceedings then terminated.



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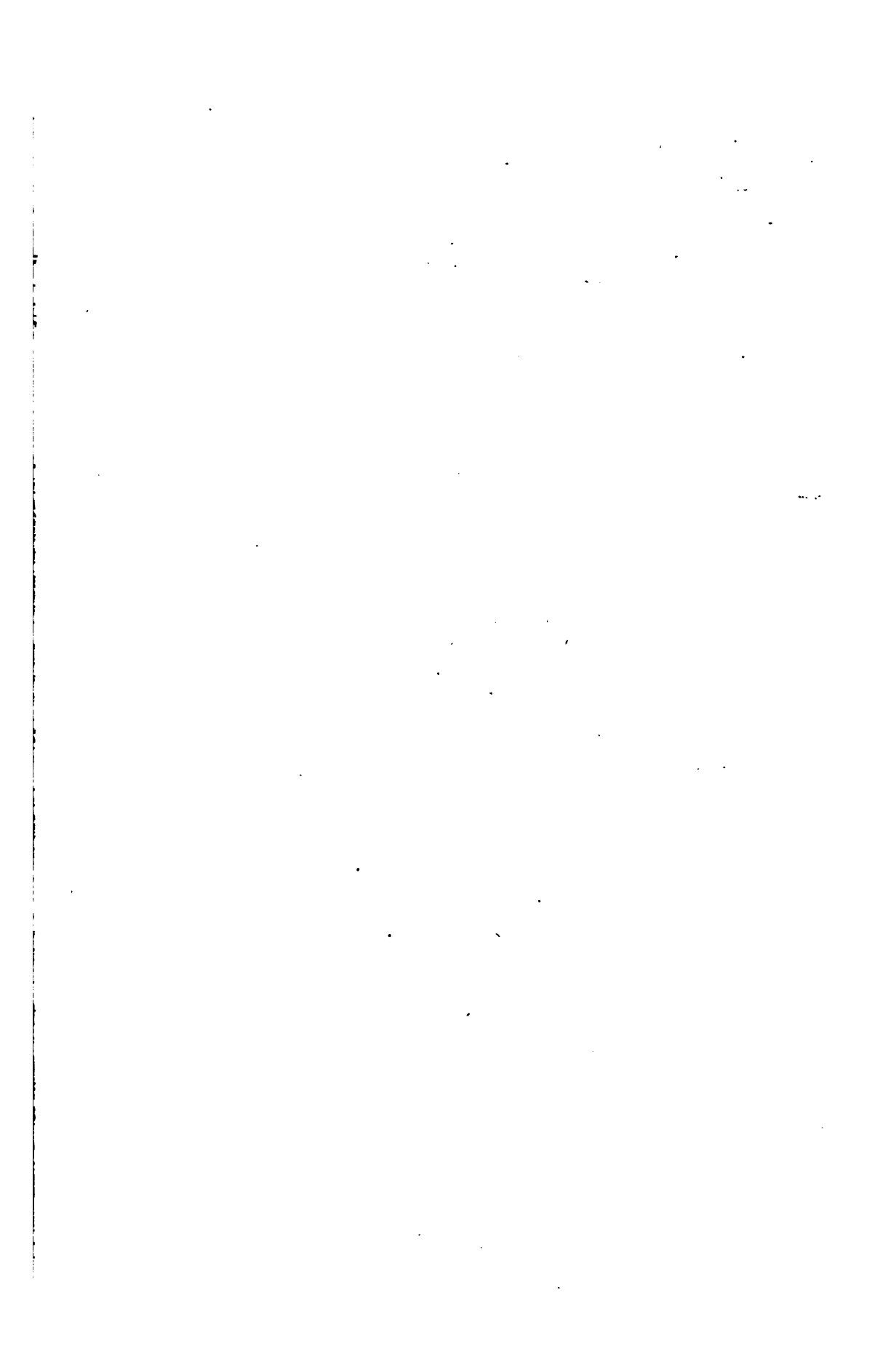
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